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NOTES ON PARASYPHILIS.[1]

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The etymology of the word syphilis is uncertain; it may be derived from the two Greek words, σῦν with, and φιλέω I love, or σῦς a pig and φίλος love, or again σῖλος, meaning "maimed" or "crippled." Any one of these derivations would be suitable to the disease, the latter perhaps the best when you come to study the far-reaching effects of the parasyphilitic affections. The word "para" is derived from the Greek παρά, meaning "beyond" or "beside," so that the term parasyphilis comes to mean something beyond or beside syphilis.

I am not quite certain in my own mind, considering we have the terms "primary," "secondary" and "tertiary" syphilis, whether it would not have been better to have adopted the less commonly used expression "quarternary" syphilis, in preference to the word parasyphilis, more especially as it has been abundantly proved of late years that there is no real distinction between syphilitic and parasyphilitic affections—spirochaetes have actually been demonstrated in parasyphilitic diseases, as described in Osler's "Principles and Practice of Medicine," in the 1916 Edition, in Wheeler's "Handbook of Medicine," 1916, and according to the Report issued by the Commonwealth Government last year on "Venereal Diseases," it is definitely stated that in the parasyphilitic conditions the spirochaetes of syphilis are still present. Sir Harry Allen, who was a most active member of that Committee, very kindly supplied me with the following references, on which this conclusion was based:—

(1) Noguchi and Moore (*Journal of Experimental Medicine*, XVII., 1913, p. 232). Treponemata found in 12 out of 70 specimens of undoubted general paralysis. A plate shows the parasite.

(2) Noguchi (*Comptes Rendus de la Société Biologique*, LXXIV., p. 349, February 15, 1913). Two new cases, sections in one packed with spironemeta, in the other spironemeta shown by ultra-microscope in fresh tissue.

(3) Forster and Tomaszewski (*Charité*, Berlin; *Deutsche Med. Woch.*, June 26, 1913, p. 1237) demonstrated spironemata in the brain after death in general paralytics. He punctured the brains of general paralytics during life and got living spironemata in two out of six cases.

(4) Marinesco and Minea (*Comptes Rendus de la Société Biologique*, LXXV., 1913, p. 231) by puncture method got the spironema in a case of juvenile general paralysis.

(5) Noguchi (*Berliner Klin. Woch.*, October 13, 1913, p. 1884). 130 new cases, 48 positive.

(6) Levaditi (*Comptes Rendus de l'Académie des Sciences*, CLVII., November 10, 1913, p. 864) successfully inoculated testis of rabbit with blood from case of general paralysis.

(7) Marinesco and Jena (*Comptes Rendus de l'Académie des Sciences*, CLIX., July 20, 1914, p. 28) did the same with cerebro-spinal fluid.

(8) Wile (*Journal of Experimental Medicine*, XXIII., 1916) took cerebral substance from general paralytics by trephining. In five out of six specimens he recognized treponemata with the ultra-microscope. He successfully inoculated the material into the testis of a rabbit. Before these important discoveries were made, however, everything pointed to the presence of the organism, such as the great majority of all general paralytics (but not tabetics) showing a positive Wassermann's reaction, and the inability to inoculate patients suffering from general paralysis with fresh syphilis.

Years before the discovery of the Wassermann reaction the acute clinical observers had demonstrated the close relationship existing between syphilis, general paralysis of the insane, and *tabes dorsalis;* but the addition of a positive reaction in such cases constitutes a definite biological bond between the early syphilitic infection and the late nervous manifestations. It must be distinctly remembered that syphilis is not a blood disease, like relapsing fever, but a definite tissue disease, although the spirochaetes are borne by the blood.

This opens up the great question as to whether the parasyphilitic affections are produced (1) by the organisms themselves, or (2) by some toxin manufactured by them, or (3) by some change which is brought about in the neurones by the spirochaetal poison, enabling a later toxin to bring about their degeneration or destruction. Mott considers that there is a primary neuronic decay, which cannot be accounted for by the changes in the supporting, enclosing, and nutrient tissues. He considers that the pathology of parasyphilitic affections is that in certain acquired or congenital syphilitic individuals the durability of the neurones is greatly curtailed, so that they decay and die prematurely, thereby giving rise to a series of symptoms which may be associated with the irritation of definite nerve structures, *e.g.*, lightening pains, visceral crises, mania, epileptiform convulsions or with neural destruction, *e.g.*, ataxy, paraesthesia, anæsthesia, paresis or dementia. There is supposed to be a difference in the strain of spirochaetes, some seem to attack chiefly bones and viscera and others the central nervous system.

The number of conditions put down to parasyphilis seems to vary with the authors' inclinations, but general paralysis of the insane, *tabes dorsalis*, and sacculated aneurysms are generally termed parasyphilitic, and sometimes a form of epilepsy, meningitis and syphilitic anæmia are included under this heading. As a digression I would like to say here that the term *tabes dorsalis* is preferable to the more commonly used term locomotor ataxy, as you can have undoubted cases of tabes without any ataxy

[1] Read at a Meeting of the South Australian Branch of the British Medical Association on August 30, 1917.

being present, so that the latter word is really a misnomer.

One of the reasons for my inflicting this paper upon you, is that I read in a book the other day how unusual it is for a practitioner who attended a patient with his primary syphilis, to see him some years afterwards when he was developing his parasyphilitic affections, it being uncommon for various reasons for the patient and the practitioner to be in touch with one another for so long a time as twenty years, but as one of my patients has been under my immediate observation for more than forty years, I thought it might be of interest to you to hear his history.

When I was articled to a firm of general practitioners in England in 1877, a gentleman came to consult them about painful lumps on his skull. As one of the firm had already attended him some six months previously for a Hunterian chancre, there was no doubt about these swellings being syphilitic nodes. The condition of the skull caused much amusement to both the doctors and the patient, and I was called into the consulting room to participate in the fun and observe my first case of cranial nodes. Iodide of potassium soon cleared up the trouble, and on their disappearance no further treatment was adopted. The patient remained in good health for some years; in 1885 he developed an extraordinary anæmia, becoming extremely pale about his mucous membranes, although his general appearance presented a most peculiar muddy pallor, quite unlike any ordinary anæmia, which I have since learnt to be typical of a syphilitic anæmia. The condition was, of course, put down to every cause except the right one, and the patient was recommended a long sea voyage, which fortunately proved effective. Good health existed till 1894, that is about 17 years after the primary infection, when suddenly after a long railway journey, ptosis, double vision, and a dilated pupil came on, and an ophthalmic surgeon was consulted. The symptoms were ascribed to rheumatism, and no enquiries were ever made about luetic disease, and the right third nerve paralysis remained for the rest of the patient's life. This did not, except for the æsthetic condition, cause much annoyance, as always afterwards the right eye was used for distance and the left for near work, and, as so frequently happens in such cases, no optical assistance was ever required. In the summer of 1896, feeling extremely fit, he thought he would take his annual holiday as a walking tour, and for a month averaged 20 miles a day, but on returning to his office it was noticed that he was unsteady in his gait, and was advised to consult his medical adviser (not the same men, be it noted, who had treated him in the early stages of his complaint), who at once recognized that with his third nerve paralysis, marked Rombergism, lightening pains and loss of patellar reflexes the case was undoubtedly one of *tabes dorsalis*. Owing to the third nerve paralysis no Argyle-Robertson phenomenon could be obtained, and there were never any signs of optic atrophy. The patient died a few weeks ago, and therefore had his tabes for 22 years, many years beyond the average life of such cases. During this period he had all the typical symptoms of the disease, and was practically bedridden for the last five years of his existence.

In 1900 he fell one morning in his bath, but as his sensory nerves were below par, he took no notice of his fall, till he began to lose the use of his right arm, and when he was stripped to discover the cause, it was at once seen that a swelling in the region of his right clavicle, evidently due to callus from a fractured bone, was pressing on the nerves of his brachial plexus. The patient himself was as much surprised as everyone else to think that he had met with so severe an accident as a broken collar bone without his being even aware of the fact. After the arm had been fixed for some weeks, the abundant callus was absorbed and the threatened brachial paralysis passed away and no further trouble from this condition was noted.

On getting out of bed one morning in 1910 his right leg gave way under him, and his right knee joint became suddenly and painlessly enormously distended with fluid. This was at first supposed to be a Charcot's knee, but after an X-ray photograph had been taken, it was found that the tibia was transversely fractured just below the joint, and the fluid was probably blood in the cavity.

It is a well recognized fact that the bones in cases of *tabes dorsalis* are extremely rarefied and fracture with the greatest ease. After some weeks the fluid disappeared, and the fracture united with an unusual amount of callus, but the leg was never of much use for locomotion afterwards.

Whilst he was in bed with the broken leg, it was noticed that the bladder had become very much distended, with a constant overflow of urine. No catheter was ever passed, as the condition caused no pain, and it is an extremely easy, but a very serious matter to infect the bladder in such cases. The *sphincter ani* became paralysed at the same time as the bladder.

About this time peculiar trophic sores began to form on the buttocks and the back of the sacrum. I have never seen their like before, heaped up circular masses of epithelium, with a central discharging crater. They would form in an extraordinarily acute fashion, the outbursts would remain for a few weeks and then disappear almost as quickly as they had come, leaving a small flat scar behind them. They reminded me more than anything of the volcanic craters you see just south of Auckland in the North Island of New Zealand, where there are some 70 or 80 small extinct volcanoes within a few square miles. The patient must have had dozens of outbursts of these; he called them his "flowerettes." They were quite painless and healed rapidly, when kept dry and well powdered with sterilized boracic acid powder.

Lightening pains in the lower limbs were excessively troublesome, they always seemed to shoot down the crests of the tibiæ, and were only relieved by migralgin; nothing else seemed to have the least effect upon them, so that when war broke out and migralgin was difficult to procure, he sent to London for 2¼ kilograms (5 lbs.), which he got at double the usual price.

About four years ago he had his first epileptiform seizure, which left him slightly aphasic for a few hours, but he never knew he had the attacks and died in one a few weeks ago. He was always of a cheerful

disposition except on east wind days, when his lightening pains were more severe. He often used to say to me how thankful he was to be afflicted with tabes and not with a cancer or some other horrible disease.

In contrast to this case I should like to mention a friend of mine who, the day he got his Commission in the Army contracted a Hunterian chancre. He went through a very severe attack of syphilis in 1879 and 1880, and wrote and told me he did not think he had a single hair left on his body. He has spent a very active life, going through three strenuous campaigns, and at the present time divides his days between looking after lines of communication on the Continent and taking a prominent position in England. He wrote me the other day that he was in perfect health, and never so hard worked in his life before. It is difficult to understand why one man who developed his disease in 1877 should die of *tabes dorsalis* in 1917, and the other, who developed his disease in 1878, should be in apparent perfect health now. It may be due to a different strain of spirochaete, or that the man who entered the army, underwent a better and more thorough form of treatment than the man who depended entirely on civilian doctors for his supposed cure. There is in all probability some peculiarity about the spirochætal poisoning which we do not understand. I had hoped to have been able to have followed up some cases of syphilis of which I had known their origin, but unfortunately the lady herself is dead and the persons she infected are now scattered broadcast over the surface of the earth. There is something subtle about the infection, as you will find that men who have been infected from the same source about the same time, go through practically the same disease and die near one another of the same parasyphilitic conditions. The wives, if the men marry, often have symptoms exactly similar to one another.

One peculiarity about parasyphilis is the rarity of *tabes dorsalis* or general paralysis of the insane in women. I do not remember ever having seen a case of general paralysis in a female, but I have seen two cases of *tabes dorsalis*. Men are far more often affected with tabes than women, whereas syphilitic disease of the rectum is almost entirely confined to the female sex, and syphilitic keratitis is far more common in female than in male children. This shows how little we understand the causes at work in the sex-distribution of disease. General paralysis of the insane is an extremely rare disease in women, and there is a theory that the toxin, or whatever it is, that affects the nervous system, attacks the part in which there is most activity going on at the time. Supposing a man is using his brain more than his limbs, he is more liable to develope general paralysis, and in the case I quoted at some length the tabes came on after a walking tour, and it is further stated that men who use their arms more than their legs, such as carpenters, are more liable to get their ataxy further advanced in their upper than in their lower limbs.

When I was a student I was always taught to be on the lookout for luetic affections in females hailing from Cathedral or barrack towns, and one of my female tabetics was born and brought up in a barrack town in England, and admitted having had syphilis as a young girl. She married out here, had no pregnancy, and about 20 years after her primary infection became a confirmed ataxic. The other female tabetic has had several miscarriages, and after a prolonged course of treatment one full-term healthy child. Although suffering from early tabes she had a fairly normal labour, and is quite ignorant of the cause of her peculiar gait, but her husband admits his infection.

So far I have been able to trace only one of my early syphilitics from their initial infection to a general paralysis, although I have no doubt in my own mind that 'ere this several of them have joined the "great majority" through this route.

Aortitis, valvulitis and cardio-vascular degenerations can hardly be classified as quarternary or parasyphilitic, as they are quite liable to come on early in the disease; but I am becoming more and more convinced that all saccular aneurysms have their origin in a syphilitic degeneration of the middle and inner coats of the main arterial system.

In the early days of my professional career I attended a youth from Port Adelaide with syphilis. He was never very severely affected, and I had forgotten all about his former illness when one day I happened to be out in the country seeing a lady, I heard a terribly loud, clangy cough come from another part of the house. On making enquiries I found it was my Port Adelaide patient, who had prematurely grown an old man, and who had been taken ill after working hard helping his gardener to sink a well. He had all the classical signs and symptoms of an aneurysm of his aortic arch, from which he died a few months after I first saw him.

I feel sure there is a quarternary condition which affects the lungs; it is very pronounced in those persons who have contracted luetic disease late in life. I can recall three such cases. In one a man contracted syphilis when over 50 years of age, and he was dead in two years. He developed a severe cough with wasting and extreme dyspnœa, but he had no tubercle bacilli in his expectoration, and his physical signs pointed more to a form of atrophic emphysema than anything else. Another patient, 20 years after his primary infection (whom I had unfortunately allowed to marry for the second time), developed a cough, increasing dyspnœa and the same apparent shrinking of the lungs without any tubercle bacilli being present. He died rather suddenly of a pneumothorax. In neither of these cases could a post-mortem examination be obtained.

The third case is that of a woman, who had a syphilitic husband and family, and whom I had explored for an inflammatory swelling in the region of McBurney's Point, which turned out to be a gumma in a Reidel's lobe of the liver. She gradually developed a cough, with great shrinkage of the lungs and increasing dyspnœa. She died of this condition, but before her end tubercle bacilli were found in her expectoration. I do not think tubercle was the primary condition of her lungs. Some of you may say that all these three cases were probably gummata at the root of the lungs, causing contraction of these organs and

hence the extreme dyspnœa, but as no autopsies were possible, the answer of this question is doubtful.

In conclusion I would like to say that I don't think that people who have had syphilis ever live to a great age. I used to think that very few ever saw 60, but the first case I have quoted lived to 62.

I am certain the older a patient is when he contracts his disease, the more malignant are its manifestations. Anyone contracting a true Hunterian chancre after the age of 45 rarely lives, as far as my experience goes, for more than five or ten years. The later in life persons become infected, the sooner the disease kills them. They seem to lose all resistance against other maladies, and die of malignant growths or tuberculosis. Two of my old male patients developed carcinoma of the penis almost before they had got over their secondary stage of the disease.

The more I see of syphilis, the more certain I become that the old saying, "that if you want to keep your syphilis, preserve it in alcohol" is absolutely correct.

ACUTE ENCEPHALITIS OF UNKNOWN ORIGIN.

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and
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I.

Clinical Report.[1]

The series of cases to which I wish to make preliminary reference to-night are, in my opinion, of sufficient importance to justify me in bringing them under the notice of members of this Branch at the earliest opportunity and inviting suggestions regarding diagnosis and treatment.

The series comprises 17 cases, all of which have been under observation and treatment at the Children's Hospital, Brisbane, since March 31, 1917. The outstanding features of the cases are these: the child, previously in good health, is suddenly seized with convulsions, which may continue, with intervals of semi-consciousness for hours or even days; subsequently the child lapses into a state of stupor, and finally deep unconsciousness. In some cases there has been a short interval of consciousness between the convulsive and stuporose stages, and one may be led to believe that the patient is recovering. In other cases the onset is more insidious, the child complaining of headache, and vomiting, convulsions supervening and the child passing into the stages of stupor and deep unconsciousness. The respirations during the stuporose stage may be loud and stertorous or grunting, but during the last stage become very slow and shallow. In one case respiration ceased, but recommenced after the application of artificial respiration for a short period; in another case artificial respiration was kept up for two hours until the heart failed. In a few cases there was head-retraction; in two cases only was there definite opisthotonus. At one time a fine tremor would be observed in a limb, for example, an arm, at another a slow rhythmic movement of, perhaps, the face and arm. Some patients, in addition, exhibited a slow, purposeless, wandering movement of the limbs, especially the arms. Nystagmus was present in one case in which the patient lived for 23 days after the onset of the illness, and was of a lightning character, the eyes, after wandering about in an inco-ordinate manner, would suddenly oscillate rapidly from side to side. The conjunctival reflex was present, and the pupils reacted to light, even in the late stages of the disease. The limbs in all cases were spastic during the greater part of the illness, flaccidity developing only in the late stages. The knee jerks were markedly exaggerated even in the flaccid stage. Babinski's sign was present in some, but not definite in others. Kernig's sign was absent. In two cases only was there a rash. In one it was of a mottled nature with underlying œdema, and in the other it was of an erythematous nature, probably due to the serum injection. All the patients had temperatures ranging from 39.4° to 41.1° C. (103°-106° F.), and the pulse was correspondingly rapid. Constipation was present in all cases. A few of the patients had nasal discharge, and a number bronchitis quite early in the disease. The duration of the illness in fatal cases was from 3½ to 23 days. All the patients except three were males. The ages varied from 1 year 9 months to 9 years. The districts from which the patients came included most of the suburbs of Brisbane. In three instances there were two cases from one family, one child falling ill within a day or two of the other. The mortality was 65%. One case is still in an acute stage with high temperature and delirium, general muscular rigidity being present. Of the six patients now convalescent, three have not regained normal intelligence. One boy has a recurrence of broncho-pneumonia, which in his case was present on admission. Only one patient appears to be making a good recovery. Some of the cases were sent in with a diagnosis of pneumonia, others of meningitis.

On lumbar puncture most of the cases showed some increased pressure of cerebro-spinal fluid, at any rate at the beginning. The fluid was quite clear, and after centrifugalizing showed on microscopic examination only one or two cells to the field in almost all cases. The globulin test applied in a few cases was negative, and the fluid did not reduce Fehling's solution. In no case have we or Dr. Harris, of the Bacteriological Laboratory, been able to grow any organism from the fluid obtained by lumbar puncture, although special media have been used and repeated lumbar punctures made. In one case, from the fluid of the lateral ventricles obtained post-mortem, Dr. Harris grew a streptococcus. In regard to the blood, in three of the cases in which a leucocyte count was made, there was a definite leucocytosis of from 17,000 to 27,000; the differential count showed polymorpho-nuclear neutrophiles 55% to 63%, small lymphocytes 21% to

[1] Read at a Meeting of the Queensland Branch of the British Medical Association on May 4, 1917.

28%, large mononuclears 9% to 22%. In six of the cases a punctate basophilia was present. In two of the cases blood cultures were made, in one case with a negative result; in the other, in which the blood was taken from the right ventricle about eight hours after death (the child having died during the night), a long-chained streptococcus was isolated. The Widal reaction was negative in the two cases in which it was done. The urine was normal except for the presence of acetone, which is found in many cases in children with raised temperatures. Post-mortem examinations were done in three of the cases, and beyond some congestion of the brain, there was nothing definite to be made out. In one of the cases in which there was a history of the child having eaten some wild berries, I opened up the complete gastro-intestinal tract, and found no abnormal appearances either in the contents or in the mucosa itself. I have kept the brain of one patient with the intention of sending it to Professor Welsh for examination.

These are the facts as far as we have observed them, but we are not yet in a position to come to any definite conclusion regarding the diagnosis. For a long time we have received into hospital patients with convulsions. These cases have proved rapidly fatal, but no definite cause could be assigned even after post-mortem examination. Some of these have been regarded as cases of lead encephalitis, although in many cases no definite signs of plumbism have been present. Nevertheless we see so much lead poisoning amongst children here that we have always to exclude it. The series I am bringing under your notice to-night, however, has characteristics of its own.

In endeavouring to classify the disease, we have excluded encephalitis of septic, tubercular and specific origin, tetanus, septicæmia and acute miliary tuberculosis, none of which occur in epidemic form. Enteric fever has been excluded by the presence of the leucocytosis and the negative Widal reaction. Lead has been excluded by the presence of the high temperatures. No cases of poliomyelitis or cerebro-spinal meningitis have been seen by us during, nor for some time previous to, the epidemic, and it is incredible that an epidemic of the purely cerebral type of either of these diseases should occur. The sudden onset with high temperature suggests a bacterial infection of some kind, but we are still in search for the causal agent.

My attention has been drawn to a paper appearing in the January number of the Transactions of the Society of Tropical Medicine and Hygiene, describing "the vomiting sickness of Jamaica," which is in some respects comparable with the sickness we have described. The main difference is that the vomiting which is such a marked feature of the former, is scarcely present in the latter. The convulsions, drowsiness, and coma succeeding the initial vomiting resemble what we have described. The sickness in Jamaica has been shown by the writer to be due to the eating of *ackee*, the fruit of *Blighia sapida*. The only similar fruit we have round Brisbane is the native tamarind, and the litchi, neither of which is in fruit at present.

Before concluding I wish to draw your attention to a series of cases of a similar nature occurring in the country, chiefly in Goondiwindi. I have seen Dr. Moore, Commissioner of Public Health, who has just returned from investigating these cases. He states that he has records of 16 cases, in eight of which lumbar puncture was done and clear fluid obtained. In one case a post-mortem examination was performed, but nothing definite was found. In 13 of the cases the ages varied from six months to eight years, in three cases from 35 to 50 years. In one instance only did two cases occur in one family. In two cases diarrhœa was present, with the passage of green stools. The mortality in this group was 43%. He has come to no definite conclusion regarding the ætiology or diagnosis of these cases.

In compiling these notes I wish gratefully to acknowledge the assistance of Dr. Alexia Maclean, the Resident Medical Officer of the Children's Hospital, Brisbane, who, being in almost constant attendance on the children, has been able to supplement my own observations.

II.

Pathological Report.

The brain and a small portion of the upper cervical region of the cord of this patient were kindly handed over to us by Professor D. A. Welsh, of Sydney University, who received them from the Children's Hospital, Brisbane, for special examination. Unfortunately, the material sent did not include the cervical, lumbar and sacral regions, with spinal pia-arachnoid, ganglia or nerve roots, or specimens of muscle and nerve. Pieces were taken and placed in (1) alcohol for Nissl's method; (2) Weigert's mordants for Heller's stain for myelin sheaths; and (3) Busch's osmic acid-sodium iodate mixture for "Marchi" reaction, and into 70% alcohol for some days, and back into water and dextrin, and cut by the frozen method for Ford-Robertson's neuroglia stain and hæmatoxylin-eosin stains.

Naked-Eye Examination.

There was considerable congestion of the pia-arachnoid and a rusty appearance of the ependyma of the first, second and third, but not of the fourth ventricle. No exudation or gross signs of inflammation were noted.

Microscopical Examination.

The main features of interest found consisted of a widespread congestion of the blood vessels, especially those of smaller size, as well as an infiltration of their adventitial sheaths, with large and small mononuclear cells, including plasma cells. The infiltration was also with cells with double nuclei, but very rarely tri-partite nuclei or polymorphs. The polymorpho-nuclear cells recognized were within the lumen of the vessels. The mononuclear cells included small plasma cells and lymphocytes, compound granular cells and large cells with a large nucleus equivalent to Leyden's epithelioid cells. The adventitial sheaths were dilated and hypertrophied, as was also the pia-arachnoid over many parts of the cortex, but

it was chiefly the inner layer of the pia-arachnoid which was most infiltrated with cells; this occurred mostly in patches. In some instances the patches were situated near to the site where the pia entered the cortex, and provided an adventitial sheath to a blood vessel; in these situations the mononuclear cell infiltration could also be traced into the cortex. In addition, many capillary hæmorrhages were noted in the pia-arachnoid and beneath the ependyma of the first, second and third ventricles, and also throughout the cortex, brain stem, pons, medulla and cord. At the same time, numerous small islets of nuclei were noted, some of them being, of course, an aggregation of monuclear cells; but in places there was, in addition, proliferation of the fixed tissue cells. The majority of the islets were of a moderately large size, and were clearly seen in the convolutions of the olive among other places. These islets also occurred in the nuclei of the aqueduct, the nulcei pontis and various nuclei of the medulla, but were not so marked as in the olive and grey matter of the cord. In the cord, while the islets were most evident in the anterior cornua, at times surrounding the anterior polar cells, the other parts did not escape; they were recognized in the postero-median septum and along the course of blood vessels entering the posterior and lateral columns of the cord. It may be said here that a capillary or other blood vessel near one of these islets could nearly always be recognized. As far as could be made out, the cerebellum seemed to have escaped altogether. The nerve cells also appeared to have been little affected, even those situated in the midst of a hæmorrhage or islet of cells. Some stained diffusely, and appeared to be in an early stage of degeneration, and some were undoubtedly missing in the olive. The outstanding feature was the interstitial reaction.

The neuroglia in the first layers of the cortex were moderately proliferated in patches, and some sub-pial felting was noted. This was not so marked as is commonly found in the earliest general paralysis coming to post-mortem examination. There was a similar hypertrophy beneath the ependyma of the cerebral ventricles, but no so-called "ground glass" granulations were noted. The neuroglial hypertrophy in the deep cortex was much more marked, especially near affected blood vessels, and numerous "spider cells" were seen in these places, as in advanced general paralysis. In fact, without the evidence of the cord and the history, we should have been inclined to consider the findings quite consonant with a case of early general paralysis, although there is commonly more disorientation of the cortical nerve cells in this condition. A case of general paralysis has been recorded in which the first change was noted in the interstitial tissue in the cortex, while there was relative escape of the parenchyma and *vice versa*. There was a marked hyperplasia of the neuroglia of the cord, extremely long processes proceeding from glia cells in the white substance; while in the grey malted, especially in the anterior horns, there was not only a dense network of fine neuroglia fibres, but also numerous neuroglia cells, which shared with the mononuclear exudation cells in composing the cell islets surrounding the anterior polar cell. Sections stained according to Heller's method for myelin sheaths revealed quite normal conditions, there being no tract degeneration, nor was there any noticeable diminution of fine fibres in the anterior cornua. It must, however, be remembered with regard both to sections stained by Heller's method and to the Marchi preparations, that the best regions for noting these changes would have been the lumbar and cervical enlargements, where were not available. Similarly, Busch preparations of the upper cord for Marchi reaction revealed absolutely no degenerating myelia. No organisms of any kind were noted.

DESCRIPTION OF ILLUSTRATIONS.

A "Watson" *camera lucida*, with eyepiece 8/1 and lenses $^2/_3$, $^1/_4$, $^1/_6$ and $^1/_9$ inch respectively were used.

Fig. 1. × 20. Section from the top of the cervical region of the cord. The distribution of the patches of round-celled infiltration and nuclear proliferation is easily recognized, and while chiefly affecting the grey matter, yet the dorsal columns are affected too.

Fig. 2. × 500. A group of anterior cornual cells, mostly staining well and of normal shape; one to the left is stained diffusely and with indistinct nucleus, and another nerve cell at the bottom is embedded in round cells, and a capillary hæmorrhage from a neighbouring capillary.

Fig. 3. × 100. Partially oblique section through cerebral cortex; note the conspicuous appearance of the cortical blood vessels, not only engorged, but also with the adventitial coats infiltrated with round cells.

Fig. 4. × 100. Cerebral cortex; vertical section. Note the thickened pia-arachnoid, whose inner layers are infiltrated with rounds cells, which accompany the pia-arachnoid into the cortex, where the latter acts as the adventitial sheath of the cortical blood vessels. At the mark "a" is seen one of the numerous patches of round cells. It is not easy to say whether these cells are wandering cells or partly fixed tissue cells.

Fig. 5. × 800. Blood vessel deep in the caudate nucleus or optic thalamus. The nature of the cells in the adventitial spaces can be seen more readily; only one polymorphonuclear cell is seen; nearly all the others are mononuclear cells, although very large, while a few have two nuclei.

Fig. 6. × 50. Part of the upper olive, showing two islets of cell proliferation, both near a small capillary, and at times these islets seem to smother the special nerve cells of these bodies.

Fig. 7. × 500. Large arteriole from the pons. Its adventitial coat is well seen crowded with mononuclear cells, and there is present a small hæmorrhage, perhaps from a small *vas vasorum* on the right upper side.

Fig. 8. × 500. Neuroglial proliferation in *cortex cerebri*. In the earliest stages that general paralytics come to post-mortem examination, the neuroglia fibres are usually longer and the cells more numerous and extend deeper. Note the strong fibres coming in from the pia-arachnoid—sub-pial felting; also a little patch of neuroglia cells at the top.

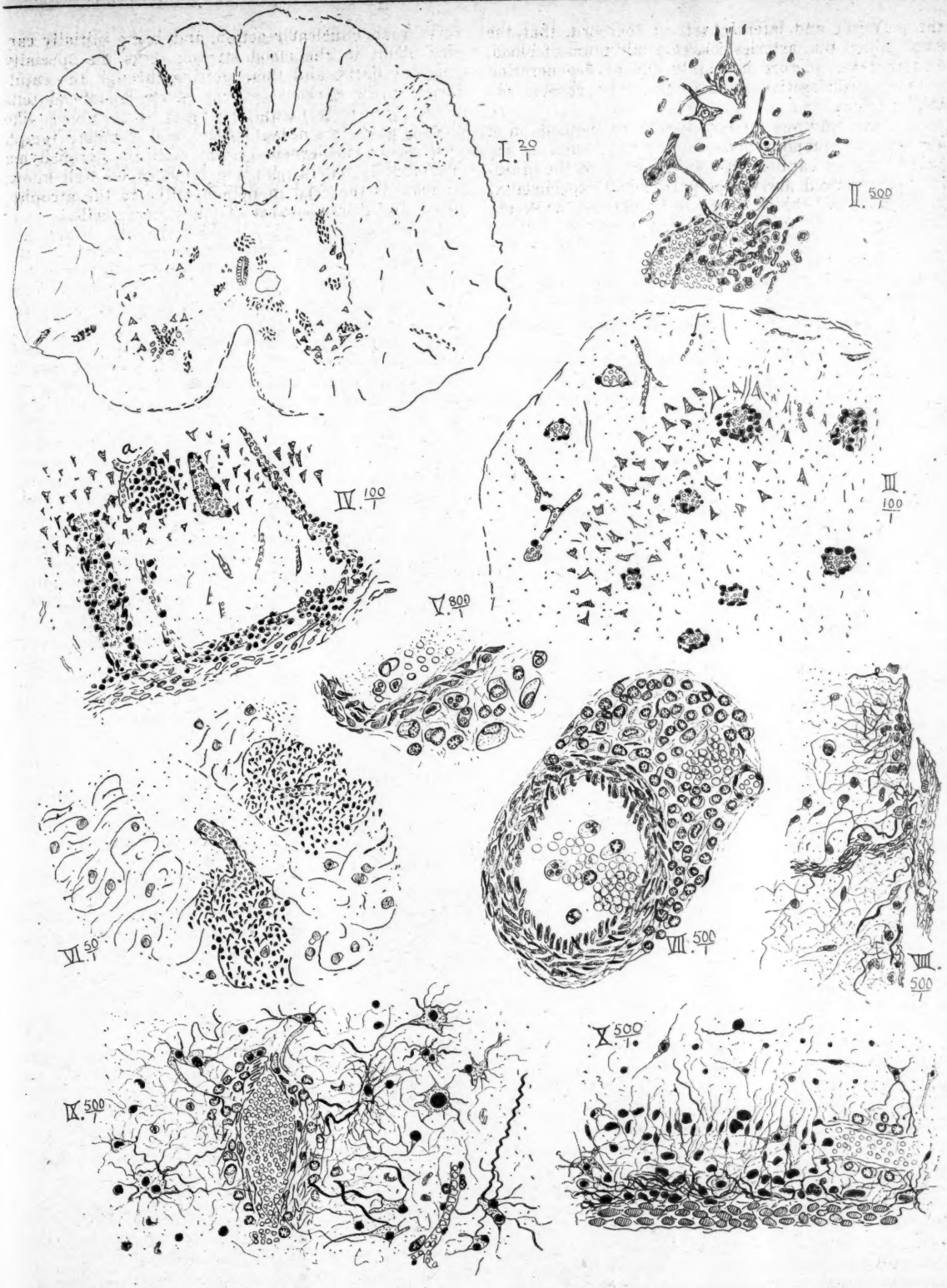
Fig. 9. × 500. Remarkable neuroglial hypertrophy and proliferation deep in the *cortex cerebri*. Apparently the fixed tissues of the part nearest the blood vessel react. They behave in the same way in general paralysis of the insane, and there often the spirochaetes are in the adventitial sheaths. Is the toxin here also produced in the adventitial sheaths? The mononuclear cells are there also.

Fig. 10. × 500. Neuroglial hypertrophy beneath the ependyma of the third ventricle; there were capillary hæmorrhages in this situation.

Interpretation.

From these pathological findings and the absence of others, can we suggest the nature of the process?

(1) Our findings do not appear to us to be due to a hæmatogenous toxin, *i.e.*, as in anæmia or pellagra, etc., in which a toxin exists in the general circulation or reaches it from some distant local, but extensive lesion, such as a peritonitis, since the researches of Orr and Rows and Williamson have shown that in these cases the brunt of the mischief falls on


I. 20/1
II. 500/1
III. 100/1
IV. 100/1
V. 800/1
VI. 50/1
VII. 500/1
VIII. 500/1
IX. 500/1
X. 500/1

the posterior and lateral parts of the cord, that the small spinal pial arteries convey a minimum of blood, and that the picture is mainly one of degeneration without proliferative phenomena. The reverse obtained in our case.

(2) Our findings do not appear to depend on a straight-out lymphogenous intoxication, such as occurs in the cord and corresponding parts of the brain-stem and brain in *tabes*, tabo-paresis and experimental bacterial toxic neuritis, since, in these cases, while the reaction is a proliferative one, yet there soon follows degenerative changes, and the brunt is still on the posterior tracts, and especially the posterior nerves and root zone. In our case the posterior tracts were not especially picked out, and the main reaction, at least in the cord, was in the very vascular anterior cornua.

(3) Septic emboli do not appear to fill the bill, because a very careful search has failed to note (*a*) a single thrombus, (*b*) a change of any kind in the lining endothelium of the blood vessels, or (*c*) a marked collection of leucocytes in the lumen of a vessel, although it may be argued that the distribution is not against this theory, affecting, as it does, the highly vascular parts.

(4) Again, cerebro-spinal meningitis chiefly affects the meninges, with signs of inflammation on the surface of the pia-arachnoid, with preponderance of polymorpho-nuclear cells, both in the meninges and during life in the cerebro-spinal fluid. The main damage in the nerve tissue lies close to the surface. In our case the main cell infiltration was mononuclear, and situated deep in the tissues. Still, among over a thousand clinical cases of polio-encephalitis observed in New York recently, 2½% showed polymorpho-nuclear cells in excess, and after an intrathecal injection of some cured patient's serum, the cerebro-spinal fluid frequently became cloudy, and over 95% of the cells contained were polymorpho-nuclear.

As we have said, the main reaction was either in the adventitial sheath, or in the central nervous system; it was nearly always very close to and associated with an adventitial sheath of a blood vessel, while the neuroglial reaction was most marked in this situation. Does not this suggest that the place where the toxin was manufactured was the adventitial sheath, or the nerve tissue in close proximity, and deeper parts of the pia-arachnoid? And in this latter connexion we are taught that as the arterioles of the pia-arachnoid have an extremely thin and inconspicuous adventitia, the trabeculæ and lymph spaces of this membrane practically form for its smaller blood vessels a common adventitia, and in support of this view is the fact that the trabecular tissue is directly continuous with the relatively very thick adventitial coats of the cortical arterioles. As stated above, the reactions so resembled those obtaining in early general paralysis that a reference to the ætiology of general paralysis of the insane might throw some light on this case. We are told that, in general paralysis, the adventitial sheaths are crowded with spirochaetes. By analogy, this condition might be caused by a minute germ or protozoon, which like the luetic spirochaete, calls forth a mononuclear reaction, and, being initially carried about in the blood stream, seeks the specially vascular parts, and then, getting through the capillaries of the nervous system, which Woodhead tells us are extremely permeable, takes up its abode, like the spirochaete, in the special and peculiar lymph channels of the nervous system, or in the parenchyma immediately outside, leading later, as we well know, as regards the cord in poliomyelitis, to the atrophy, death and disappearance of many nerve cells.

In poliomyelitis we get cord changes, which are usually unilateral in any one section, owing to the fact that the branches from the anterior spinal artery are usually given off alternately to either side. These changes consist of various stages of degeneration of the anterior spinal nerve cells, less frequently in the posterior horn, in the fine fibres near the cells, in anterior nerve roots, and even in isolated myelin fibres in the lateral and posterior tracts. There is excess of neuroglia in the parts affected, especially near the blood vessels, and also spider cells, including many nuclear elements. The blood vessels have their middle and external coats thickened, the latter as well as the pia-arachnoid, and the grey matter of the anterior horn being crowded with small and large round cells, including *Gitterzellen* and large epithelioid cells; there are also occasional capillary hæmorrhages. The cells are affected not necessarily in the functional groups, but only as they are supplied by one capillary or system of capillaries. The old ideas of thrombosis as a cause, or a primary degeneration of the neurone without interstitial changes, are not now held.

Finally, we have degeneration of muscle fibres and neurones secondary to affection of the cells. Rarely are similar changes to be found in the motor nuclei of the *medulla oblongata*. Redlich has recorded the occurrence of poliomyelitis and encephalitis in the same subject. Still more recent researches suggest this to be the rule. Lastly, the likeness of these lesions to those experimentally produced by bacteria is referred to. We must also bear in mind Flexner's experiments with infected monkeys.

The findings in our case from Brisbane show a close analogy to this well-known pathological syndrome. The relative escape of the nerve cells, spinal and cortical, should, however, be noted. The analogy is especially remarkable in connexion with the rarer clinical forms, such as the cortical and the non-paralytic or abortive, including the meningitic form. We must remember, too, that in many cases without evident clinical signs of encephalitis, special pathological methods reveal this condition when, at rare intervals, the specimens reach the pathologist. Finally, the examination of this case by itself strongly points to the lesions being due to an infective agent, chiefly localized in the adventitial sheaths and inner layers of the pia-arachnoid, and brought there by the blood stream.

It gives us much pleasure to acknowledge our indebtedness to Dr. A. W. Campbell and Professor D. A. Welsh for their assistance.

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Public Health.

NEW SOUTH WALES.

The following notifications have been received by the Department of Public Health, New South Wales, during the week ending October 13, 1917:—

	Metropolitan Combined District. Cs.	Dths.	Hunter River Combined District. Cs.	Dths.	Rest of State. Cs.	Dths.	Total. Cs.	Dths.
Enteric Fever	2	0	0	0	6	0	8	0
Scarlatina	10	0	2	0	4	0	16	0
Diphtheria	24	1	0	0	30	1	54	2
C'bro-Sp'l Menin.	3	0	0	0	1	0	4	0
Malaria	0	0	0	0	1	0	1	0
Pulmonary Tuberculosis*	17	9	1	0	0	0	18	9

* Notifiable only in the Metropolitan and Hunter River Districts, and, since October 2, 1916, in the Blue Mountain Shire and Katoomba Municipality.

VICTORIA.

The following notifications have been received by the Department of Public Health, Victoria, during the week ending October 14, 1917:—

	Metropolitan. Cs.	Dths.	Rest of State. Cs.	Dths.	Total. Cs.	Dths.
Diphtheria	40	0	22	0	62	0
Scarlatina	28	2	16	0	44	2
Enteric Fever	1	0	1	0	2	0
Pulmonary Tuberculosis	20	9	7	0	27	9
C'bro-spinal Meningitis	3	—	1	—	4	—

QUEENSLAND.

The following notifications have been received by the Department of Public Health, Queenslanad, during the week ending October 13, 1917:—

Disease.	No. of Cases.
Diphtheria	29
Pulmonary Tuberculosis	9
Scarlatina	3
Enteric Fever	5
Malaria	1
Ankylostomiasis	1
Erysipelas	1

SOUTH AUSTRALIA.

The following notifications have been received by the Central Board of Health, Adelaide, during the week ending October 6, 1917:—

	Adelaide. Cs.	Dths.	Rest of State. Cs.	Dths.	Totals. Cs.	Dths.
Diphtheria	1	1	20	1	21	2
Pulmonary Tuberculosis	4	0	13	4	17	4
Pertussis	0	0	6	0	6	0
Morbilli	0	0	4	0	4	0
Scarlatina	0	0	4	0	4	0
Enteric Fever	0	0	4	0	4	0
Erysipelas	0	1	2	0	2	1

TASMANIA.

The following notifications have been received by the Department of Public Health during the fortnight ending October 13, 1917:—

Disease.	Hobart. Cases.	Launceston. Cases.	Country. Cases.	Whole State. Cases.
Diphtheria	5	3	33	41
Pulmonary Tuberculosis	2	1	2	5

WESTERN AUSTRALIA.

The following notifications have been received by the Department of Public Health during the fortnight ending October 6, 1917:—

	Metropolitan. Cases.	Rest of State. Cases.	Totals. Cases.
Enteric Fever	1	2	3
Diphtheria	10	12	22
Scarlatina	4	3	7
Pulmonary Tuberculosis	8	6	14
Erysipelas	1	2	3
Cerebro-spinal Meningitis	1	0	1

Vital Statistics.

SOUTH AUSTRALIA.

From the returns of the births and deaths registered in South Australia during the month of July, 1917, the following information has been published. There were 891 births, as compared with 1,088, which was the average for July of the years 1912-1916. The birth-rate is equivalent to an annual rate of 24.72. This is the lowest birth-rate for July for five years. In July, 1914, it was 31.8, and in the following year it was 28.08 per 1,000 of population. The number of deaths registered during the month was 366, which is equivalent to an annual death-rate of 10.2 per 1,000 of population. The rate is considerably lower than that of July of the preceding years. In 1916 it was 12.36. Of the 366 persons who died, 43 were under one year of age, and 39 were 80 or over. The infantile mortality was 48.2 per 1,000 births.

Among the causes of death, diseases of the cardio-vascular system were held responsible in 62 instances, in 39 there was organic disease of the heart, in 22 there was cerebral hæmorrhage, and in one there was acute endocarditis. Of the infective processes, tuberculosis caused 26 deaths, pneumonia 22, diphtheria 10, bronchitis 11, diarrhœa and enteritis 5, acute nephritis 2, and enteric fever, non-puerperal septicæmia, simple meningitis and puerperal septicæmia one each. There were 32 deaths from malignant disease, 21 from Bright's disease and 11 from diabetes.

The number of births registered in the city of Adelaide was 72, which is equivalent to an annual birth-rate of 21.84. This rate is considerably lower than the average for the corresponding month in the preceding five years. The number of deaths, excluding those which took place in public institutions of persons not usually resident in the city, was 51, and the death-rate was equivalent to an annual rate of 15.12 per 1,000 of population.

TASMANIA.

The Government Statistician has published the vital statistics of the urban and of the rural districts of Tasmania for the month of July, 1917. The births registered in Hobart during the month numbered 117, and in Launceston 66, making a total of 183. Of these, 89 were males and 94 females. While this number is slightly larger than the number of births registered in July, 1916, it is somewhat smaller than the average for the corresponding month of the preceding five years. The birth-rates, expressed as annual rates, were 35.4 in Hobart, 32.28 in Launceston, and 34.2 in the combined urban districts.

The number of deaths registered in the urban districts was 93, 57 being in Hobart and 36 in Launceston. Of the persons who died, 53 were males and 40 females, while in 29 instances the death occurred in public institutions. The

death-rate, expressed as an annual rate, was 17.28 in Hobart, 17.52 in Launceston, and 17.4 in the urban districts. The Statistician calls attention to the fact that 89 of the 93 deaths actually took place in July. The corrected death-rate is not given.

There were 17 deaths due to diseases of the cardio-vascular system, 30 of which took place in Hobart and 4 in Launceston. Tuberculosis was responsible for seven deaths (four in Hobart and three in Launceston). There were three deaths from pneumonia in Launceston and one in Hobart. There were two deaths from acute nephritis in Hobart and one in Launceston, and there was one death from pertussis in Hobart, one death from influenza in Hobart, one death from simple meningitis in Hobart, one death from diphtheria in Launceston and one from acute bronchitis in Launceston. There were five deaths from cancer in Hobart and four in Launceston, and three deaths from Bright's disease in Hobart and two in Launceston.

The total number of births registered in the country districts during the month was 282. This figure is lower than the figure for July, 1916, and higher than the figure for July, 1916. The death-rate, expressed as an annual rate, was 24.96 per 1,000 of population. The total number of deaths registered was 63. The death-rate was consequently 5.52, which compares favourably with the equivalent death-rate in July, 1915, and July, 1916, when the rates were 9.2 and 6.96 respectively.

Five of the deaths were due to tuberculosis, four to malignant disease and two to diphtheria. The Statistician gives a few details concerning the causes of death under the heading of "General Diseases," while he groups all the other diseases together without further enumeration.

CONTRACT PRACTICE IN VICTORIA.

Points in Organization.

III.

The Remuneration.

The Common Form of Agreement which the Council of the Victorian Branch of the British Medical Association has drawn up for universal use in the State, provides for a *per capita* rate of remuneration of not less than 20s. per member in Melbourne, Ballaarat and Bendigo, and not less than 26s. per member elsewhere. In New South Wales the rates fixed by the approved Common Form of Agreement are as follows: In Sydney and suburbs the minimum rate is 21s. A few Lodges accepted the Common Form of Agreement before June, 1914, on the understanding that the rate of 19s. should be recognized as a minimum rate. These rates do not include the provision of medicine. Elsewhere the minimum rate without medicine is 26s and with medicine 34s. Where higher rates were in operation at the time of the introduction of the Common Form of Agreement, these higher rates have been accepted as the minimum rates for the districts concerned. In Great Britain the rate under the National Insurance Act is 7s. per person, or 28s. per family, exclusive of medicine. It is assumed that the average number of members of a family is four.

Hitherto the rates paid in the suburbs of Melbourne have varied considerably. In Malvern the majority of the Lodges have adopted the rate of 15s., in Carlton the rate is 12s. 6d., and in the other suburbs it averages 14s. In some instances a different rate is paid by members of the same order but different Lodges, while in other cases the rate is not uniform, even in the same suburb. The Manchester Unity Independent Order of Oddfellows pay 12s. 6d. in Collingwood, while in Richmond one Lodge pays 16s. and another 15s. A Lodge in Melbourne pays 30s. per annum, but the majority of its members have incomes considerably above the limit imposed by the "Income Limit" clause.

The rates paid in Lodges which have female members also vary. In Port Melbourne the rate is the same as that for male members, *i.e.*, 14s. per annum. In some of the Lodges the rate for female members is as low as 7s. 6d. a year.

In the country the rate of 17s. is paid in some of the Lodges, while the sum of 42s. is paid in one district, where the work is certainly not harder than elsewhere. The Council is insisting that the minimum rate in the country districts shall not be less than that obtaining at present. The rates of 20s. and 26s. have been fixed as the minimum rates, and where more is being paid, the higher rate will continue to be claimed.

When a medical practitioner receives an agreement for signature, it will be necessary for him to examine the remuneration clause, in order that a pre-existing higher rate is not reduced. In the event that the Lodge has been paying more than 26s. the words "minimum rate of 26s." must be struck out and the older, higher rate substituted. In these circumstances the practitioners in the district will find it advisable to hold a meeting for the purpose of ascertaining the prevailing rate and of arranging that the agreements shall be uniform.

The rate of remuneration was fixed before the outbreak of war. The President of the Friendly Societies' Association recently stated that the cost of living has increased by about 50% since June, 1914. It has been shown that the cost of living has increased by 100% since the bank crashes, 25 years ago. It was prior to this immense increase in the cost of the necessities of life that the present rate of remuneration for the Lodge surgeons was agreed to. In the interval the wage-earners have insisted on repeated increases in their wages in order that they might be able to meet the growing expenses of food and house rent. The Lodge surgeon has been required to work for the same inadequate fee, despite the increased cost of living and despite the increases in wages. The wage-earners have received protection by the Factories Acts and the Arbitration Courts. Working hours have been shortened, wages have been increased and the cost of production has gone up; the employers have passed the burden on to the consumer. It may be argued that the demand for higher wages has scarcely benefited the worker, since prices have gone up to enable the employer to pay the increased wages. On the other hand, it must be conceded that the workers are responsible for a part, at all events, of this state of affairs. The medical officers of the lodges have to meet the added cost of living without any compensating increase in remuneration to ease the burden. He is now compelled to travel in motor cars, which are much more expensive than horse and trap. The modern methods of diagnosis and treatment are more costly than those which were employed exclusively a quarter of a century ago. Servants' wages, the price of petrol and taxes are special instances of increased expenditure which the doctor finds difficult in paying for out of Lodge remuneration. In the last place, some of the members of Friendly Society Lodges are not wage-earners, but are engaged in farming and other occupations which have proved highly productive within the past twenty years.

The increase which the medical profession is demanding of Lodge patients is from 3½d. to 5d. a week. From the figures quoted above it will be seen that the Victorian doctors have fixed their Lodge rates at a somewhat lower scale than the New South Wales doctors did in 1914. In our opinion this action is unfortunate, but there is at present no escape from the position. The Victorian Branch has offered this rate, and for the present, it must adhere to it.

Since the wage-earners have obtained their demands by combination and mutual aid, it would seem that the same means must be employed by medical practitioners. Unity is essential. The Lodge patients will not be left without medical attention, because of the united action of the doctors. They will be able to obtain medical attendance when they fall ill, but until the Lodges accept the terms of the Common Form of Agreement, the arrangement between doctor and patient will have to be a private one and not one dictated by officials and an executive.

AUSTRALIAN ARMY MEDICAL CORPS COMFORTS FUND.

We have pleasure in thanking two contributors to our appeals for funds to enable the Australian Army Medical Corps Comforts Fund Depôt to carry on their fine work. The total amount received through this journal is £18 17s. short of £100. Although we are asking for a larger sum than £100, we trust that members will enable us to pass this mark during the next week or two.

	£	s.	d.
Amount previously acknowledged	78	0	0
Dr. W. W. Martin (Wagga Wagga, N.S.W.)	1	1	0
Dr. John Higgins (Clermont, Q.)	2	2	0

The Medical Journal of Australia.

SATURDAY, OCTOBER 27, 1917.

A Duty—Not a Burden

The delegates of the local medical association of New South Wales discussed at their annual conference with the Council of the New South Wales Branch of the British Medical Association the best means of assisting the widows of our gallant colleagues who give their lives for our safety and the families of those who suffer irreparable harm in the exercise of their duty as medical officers in His Majesty's sea and land forces. No definite conclusion was arrived at, but the majority favoured the proposal that those who remain at home, should bear the cost of insuring the lives of those who are on active service. This subject has claimed our attention on several occasions in the past. Dr. F. S. Hone, of Adelaide, first suggested that something should be done, and invited the opinions of others whether that something should be a combined action of the medical profession in the Commonwealth or whether it should be undertaken by the several Branches of the Association. At the annual meeting of the South Australian Branch, held on June 28, 1917. Dr. J. C. Verco included this subject in his excellent Presidential address and made some very practical suggestions. He pointed out that any money that was given to a fund instituted for this purpose, would not be given as a charity or as a recompense, but as a willing thanksgiving to men who richly deserve all we can give and more. It would seem that, notwithstanding the fact that the members of the medical profession in Australia approve of the principle of the proposal, many feel that the undertaking would be too large an one for them to bear, and consequently the matter has been allowed to drift. It is true that in not a few individual cases insurance policies have been taken out by one or more of the personal friends of men departing for the front, and these policies have been kept effective. The task of making adequate provision for those in need of it, however, is not so vast as it would appear at first. In the first place, it is probably unnecessary to make any special provision for the men who are sent to base hospitals or other places of comparative safety. The risk attached to work in military hospitals is not materially greater than that of civil hospital practice. In the next place, practitioners who have established themselves well and have good practices, senior members of the profession, and those with well-earned reputations will probably not need assistance. In the third place, the young, unmarried men without dependants, would only require help if incapacitated for a time or permanently. The insurance problem can, therefore, be narrowed down to a scheme for taking out policies for men early in their careers, who have wives or other dependants and who are engaged in or near the firing line. The number of these men must be relatively small. It should be an easy matter for the doctors in the Commonwealth to provide the premiums for policies to insure fifty young men for a thousand pounds each. In addition a sum of money should be available for the purpose of giving support to those men who return to the Commonwealth, disabled to a greater or lesser extent, and to those who find that a promising practice has dissipated into thin air. A matter of greater difficulty is that of discovering what actual provision should be made and to whom the aid should be offered. If the movement is to be Federal, and we are strongly of opinion that the artificial barriers of States should be broken down in this instance, it would be essential for each Branch of the Association to set up local committees to administer the fund locally and to undertake the task of advising the Federal Committee of the Branches of the British Medical Association in Australia as to what provision would have to be made. Energy and activity would be required of the Federal Committee, and the members would have to sacrifice time to perform the co-ordinating part of the work. Unfortunately the machinery of this Committee is clumsy, and it takes weeks and longer to set the wheels in motion. It might, therefore, be advisable if the several Branches would consider simultaneously whether some immediate action could not be taken, so that at the next meeting of the Federal Committee in February, 1918, working propositions could be handed over to it to co-ordinate and adjust. Dr. Verco is of opinion that three thousand pounds could be collected

in South Australia. Surely the other Branches could secure substantial sums of money, so that the fear of failure would have disappeared before the February meeting. The success of an undertaking of this kind would depend on the recognition of each member of his obligation to his colleagues in Flanders and elsewhere in the war zone.

THE MILK SUPPLY OF SYDNEY.

The milk for the city of Sydney is derived from cows kept by suburban dairymen and from herds owned by farmers living in the country at variable distances from the metropolis. The milk sent by the farmers to Sydney is distributed by several companies which handle roughly two-thirds of the daily supply. Enumeration of the bacteria present in the milk delivered to households in the morning shows that the milk contains several thousand microbes in each cubic centimetre when it comes from a local dairy, but that the milk harbours several million bacteria in each cubic centimetre when it is received through the distributing companies. Although these two varieties of milk differ so greatly in regard to contamination with bacteria, it is obvious that the country milk at one time contained as few germs as the suburban milk. The bacteria in the country milk on its delivery to the consumer are produced by a thousand-fold multiplication of the microbes which have accidentally fallen into it. If the suburban milk was kept before delivery for as many hours as the country milk, the number of bacteria present would be similar.

It is well known that the sour curdling of milk is brought about by ferments formed by micro-organisms. Unless the microbes multiply in the milk, lactic acid is not produced from the sugar of milk in sufficient amount to precipitate the caseinogen. In like manner the putrefactive and other fermentative processes which render milk unfit for human use, are dependent on the growth of different kinds of germs in the milk. The speed with which milk is altered by chemical changes, is determined by the number of bacteria engaged in manufacturing the ferments which act on the various constituents of the milk, and by the temperature at which these enzymes operate. Country milk does not exhibit changes due to the growth of bacteria for twelve hours, even when it is kept at summer heat (30° C.).[1] At lower temperatures the chemical transformations are delayed for longer periods.

The companies distributing milk are desirous of selling as pure an article as possible. They endeavour to limit the number of germs in the milk by imposing strict rules for milking the cows and by paying attention to the cleanliness of the cans in which the milk is forwarded to Sydney. The conditions of supply and distribution make it, however, impossible to deliver milk less than twenty-four hours old. In this time the bacteria which enter the milk despite all the precautions to ensure purity, would multiply to such an extent that fermentative changes would render the milk sour, if steps were not taken to reduce the bacterial contamination. The milk is heated in Sydney to kill the microbes and is cooled after pasteurization. These processes are, however, not controlled by adequate scientific methods. An engineer is responsible for the working of the pasteurizing and cooling plants and a chemist tests the milk for adulteration with water, but no bacteriologist examines the milk to ascertain how many bacteria are present in the milk. Pasteurizing plants require constant care if they are to do their work satisfactorily. It has been often noted that the number of germs in the milk at the end of the process has been no less than at the beginning. The microbes that are already in the milk, are destroyed in the heating tank, but the milk is contaminated again in the cooling coils. Unless examinations are made from time to time of the bacterial content of the milk during the stages of cooling the heated milk, pasteurization may do little more than change the bacterial flora of the milk. In this connexion it may be noted that dairy or farm milk is always infected with germs forming lactic acid from lactose, but that pasteurized milk may be only infected with putrefactive organisms. The unheated milk will become sour, thus warning the consumer of its age, while the putrefied milk may show no sign to the sense indicating its toxic nature. A biochemist in the employ of the milk companies, should be responsible for the correct use of the pasteurizing plant, as well as for the composition of the milk.

In common with other physiological processes the reproduction of micro-organisms is governed by temperature. The rate of growth of bacteria is lessened as the temperature falls. Cooled below 5° C. (40° F.) fresh milk keeps for many days, if not indefinitely. A temperature of 10° C. (50° F.) will prevent the multiplication of microbes for forty-eight hours. Even at 15° C. (60° F.) bacteria multiply slowly in milk. Not only will a low temperature retard the growth of microbes, but it inhibits the activity of bacterial enzymes. A milk containing several million organisms per cubic centimetre may keep well at a low temperature, as no chemical changes can be induced in the constituents of the milk. When such a milk is warmed, it deteriorates rapidly. This behaviour is well known in Sydney to the consumer of milk from a country source. These milks will only keep sweet for a few hours on a warm day, though they will remain wholesome for many hours in wintry weather.

When it is observed that fresh country milk contains as few bacteria as the milk of suburban dairies and that the multiplication of these bacteria is easily prevented by cold, it would appear desirable to cool milk before transport to Sydney. With cold transport by rail such milk could be kept in the same condition as suburban milk. Physicists have put it on record that once a large quantity of liquid has been cooled to a low temperature, it will remain below the temperature of the air for many hours. Large tanks of cold milk will travel in a train for many hours with only a change of temperature of a few degrees. The practice of sending cold milk in bulk has been used at

[1] Chapman, Proc. Linn. Soc. New South Wales, Vol. xxxiii., p. 440, 1908.

certain stations where a large supply of milk can be collected and cooled before dispatch. The cooling of cans of milk has not yet been attempted. Conduction of heat through the air is so slow that such cans are only cooled rapidly by direct contact with a cold liquid. A design for a can holding milk which could be effectively cooled in a few minutes would be a great boon.

Milk for the metropolitan area should be cooled, if possible, on the farm. If this cannot be done, the milk should be sent at once to the local depôt, where it should be cooled to just above the freezing point. After its railway journey, for which refrigerating cars might be provided, it could be readily cooled in the tanks of the distributing company. Pasteurization on the large scale might be dispensed with. If it is required to guard against infection with pathogenic germs through the milk, the heating is best performed in the house after delivery as the last act in the history of the milk. The final sterilization would serve to prevent all infection through the milk.

THE HOOKWORM SURVEY IN QUEENSLAND.

The International Health Board of the Rockefeller Foundation recently made a proposal to the Queensland Government to organize a hookworm survey from Thursday Island to Brisbane, provided that the Queensland Government was prepared to defray approximately one-third of the expenses incurred. Estimates were drawn up and the extent of the liability was fixed at £1,552 for the Government, the Foundation agreeing to supply the remainder. The proposal included treatment of all persons found to be suffering from the infestation and a well-devised education scheme, as a part of the prophylaxis. Considerable delay occurred, which may have been due to the various other important questions with which the Government found itself faced. On October 8, 1917, Dr. Waite, who is engaged on an ankylostomiasis campaign in Papua at present, discussed with the Premier the arrangements suggested. On the following day the Home Secretary made an announcement in the press to the effect that the Government had accepted the generous proposal of the Rockefeller Foundation. We understand that the campaign will be started early in 1916.

Naval and Military.

The 342nd list of casualties, issued on October 18, the 343rd list, issued on October 19, and the 344th list, issued on October 22, 1917, contain in the aggregate the names of 2798 officers, chaplains and men. The dead number 790 and the wounded 1,940. It is with extreme regret that we learn that Captain George Stephenson Elliott was killed in action on September 25, 1917, and that Lieutenant-Colonel James Joachim Nicholas has also been killed in action. The wounded include Major George Aloysius Makinson Heydon, M.C., Major William Loderwyk Crowther, and Captain Sydney Clarence Morris. Major Crowther is reported to be remaining on duty. Captain Frank Bramall Metcalfe is reported to be dangerously ill.

From other sources we learn that Captain John Rowland Tillett has died of wounds on October 2, 1917. It is also reported that Captain Frank Bramall Metcalfe, whose name appears in the 342nd list of casualties under the heading of dangerously ill, died of shock on October 6, 1917, after the operation of amputation of his leg. It is stated that the operation was rendered necessary by a wound received while on duty. Major Gladstone Montague Hunt, M.C., was killed in action on October 4, 1917.

The following appointments have been notified in the *Commonwealth of Australia Gazette*, No. 178, under date of October 18, 1917:—

Australian Army Medical Corps.

To be Majors—

Captain J. J. McMahon, No. 10 Field Ambulance.
Captain W. R. Kelly, No. 3 Australian Sanitary Section.
Captain W. W. W. Chaplin, No. 10 Field Ambulance.
Captain H. C. E. Donovan, No. 9 Field Ambulance.
Captain A. V. Meehan, No. 9 Field Ambulance.
Captain J. S. Smyth, No. 3 Australian Casualty Clearing Station.
Captain R. I. Furber, No. 4 Field Ambulance.
Captain N. J. Bullen, No. 15 Field Ambulance.
Captain D. deC. Browning, No. 3 Australian Auxiliary Hospital.
Captain J. Kenny, No. 7 Field Ambulance.
Captain J. Macdonald, No. 2 Command Depôt.
Captain J. P. Fogarty, Australian Imperial Force Dopôts in England.
Captain J. W. Wilkinson, Australian Imperial Force Depôts in England.
Captain D. J. W. Browne, No. 12 Field Ambulance.
Captain J. C. P. Strachan, No. 16 Field Ambulance, Wareham.
Captain F. T. Wheatland, No. 10 Field Ambulance.
Captain W. B. Craig, R.M.O., 22nd Battalion.
Captain C. C. Corlis, No. 13 Field Ambulance.
Captain F. T. A. Lovegrove, No. 1 Command Depôt.
Captain A. Goldstein, No. 6 Field Ambulance.
Captain (temporary Major) T. L. Anderson, No. 3 Australian General Hospital.
Captain J. Hardie, M.C., R.M.O., 21st Battalion.
Captain H. P. Brownell, R.M.O., 27th Battalion.
Captain A. J. Kelsey, R.M.O., 25th Battalion.
Captain C. N. Smith, B.E.F., France.
Captain C. E. Marshall, No. 8 Field Ambulance.
Captain A. F. Maclure, No. 2 Australian General Hospital.
Captain R. St. C. Steuart, No. 3 Australian General Hospital.
Captain A. J. Brenan, No. 1 Australian General Hospital.
Captain L. A. Hayward, R.M.O., 1st Divisional Engineers.
Captain R. E. McClelland, No. 1 Australian General Hospital.
Captain E. H. M. Stephen, No. 1 Australian General Hospital.
Captain A. J. Dunn, No. 2 Command Depôt.

All the above dated 27th April, 1917.

Captain Arthur Duncan Forbes, Royal Army Medical Corps, is appointed Captain in the Australian Imperial Force. Dated 20th April, 1917. (This cancels the notification respecting this officer which appeared in Executive Minute 620/1917, promulgated in *Commonwealth of Australia Gazette*, No. 155, dated 13th September, 1917.)

His Excellency the Governor-General has been pleased to approve of Walter Henry Gors being granted the honorary rank of Captain (temporarily) in the Army Medical Corps Reserve, Australian Military Forces, whilst employed in connexion with the Australian Branch, British Red Cross Society. Dated, September 29, 1917.

We have been asked to state that the annual general meeting of the Sydney and Suburban Provident Medical Association will be held at the offices of the Association on the third floor of the B.M.A. Building, 30-34 Elizabeth Street, Sydney, at 8.30 p.m. on Wednesday, October 31, 1917.

Abstracts from Current Medical Literature.

MEDICINE.

(144) Pharmacology and Therapeutics of Alcohol.

Bernard Fantus, in a lecture before the Chicago Medical Society, discourses on the rôle of alcohol in the welfare of the human race (*Journ. Amer. Med. Assoc.*, July 7, 1917). He states that, in its effect on the central nervous system, alcohol is a depressant from the very beginning. It causes stimulation by reason of the fact that removal of the higher functions produces an apparent stimulation of the lower ones. It impairs and abolishes, first, the highest and last acquired functions—self-restraint, attention, observation, reflection and judgement. It enables a man to drown his sorrows, but is such a good preservative that it keeps the sorrow for the morrow. It is proved that even a small dose slows the speed and lessens the accuracy of thought; yet the person experimented on has the curious illusion that he has performed the act more quickly and better, for the finer grades of judgement have become lost. Alcohol makes a man feel strong, and yet it strengthens no one; it merely benumbs the sense of weariness. Why do human beings put this stuff into their mouths, that robs them of their brains? They do this for the same reason that human beings do anything, *viz.*, to derive pleasure or escape pain. The man who takes pride in the notion that he can drink or leave drink alone, just as he chooses, should test himself by suddenly cutting out drink. He will probably be shocked at the hold the craving for liquor has on him. It is possible for some to drink moderately during a long lifetime; but no one can tell whether or not he is one of these. Alcohol induces a feeling of warmth, while at the same time it lowers body temperature, for it dilates the blood vessels of the skin; man is accustomed to judge his temperature by the amount of blood in the skin. Freezing to death most commonly takes place in intoxicated persons. Sunstroke is generally sustained from the combined effect of liquor, hard labour and strong sunshine. Alcohol is not a true stimulant to the circulation. Stimulation of the heart does not occur under ordinary circumstances. Deep vasoconstriction and superficial vasodilation are the chief changes produced. The best use of alcohol is in shock. Here its narcotic action, which lessens the appreciation of the injury sustained, produces, no doubt, the chief benefit. Nevertheless, the reflex stimulation from the stomach is not to be despised. Alcohol is more truly a stimulant to gastric digestion than to any other function of the body. The normal stomach, however, needs no such stimulant, and the diseased stomach is not cured by it. Indeed it is the most important single cause of gastritis in the adult. Alcohol is undoubtedly a food. It has a higher caloric value than protein or carbohydrate. When, however, food is taken in abundance, it is not only superfluous as a nutriment, but is positively harmful. Habitual consumption of it, no doubt, frequently contributes to the evolution of arterio-sclerosis and of degeneration of the parenchyma and overgrowth of the connective tissue of various organs of the body, especially the liver and kidney. Alcohol cannot be considered a useful food for healthy persons. In speaking of fever, the writer observes that the rule that a narcotic drug habitué should not be deprived of the drug his system demands, when serious sickness overtakes him, should no doubt be applied here. When a drunkard has pneumonia, it is a poor time to attempt to break him of his liquor habit. A warning is given against prescribing alcohol in convalescence from acute disease, or in chronic ailments, such as insomnia. Alcohol is merely a symptom remedy. It does not cure anything except, perhaps, neuralgia of the fifth cranial nerve by direct injection (combined with cocaine) into the various branches of this nerve. Should humanity be deprived of liquor, it would lose a consoler, but would have far less need for consolation. Alcohol may afford a man a sense of well-being, but it certainly does not contribute to the welfare of the human race.

(145) Latent Syphilitic Infection of the Lungs.

H. R. M. Landis and P. A. Lewis describe five cases of pulmonary syphilis, in all of which the symptoms were strongly suggestive of pulmonary tuberculosis. These symptoms were: morning cough and expectoration, blood-streaked sputum, loss of weight and slight elevation of temperature. In two there was pain resembling that of pleurisy at the base of the right lung. Further, all the patients had physical signs of incipient tuberculosis (*XIII. Report of Henry Phipps Institute*, Philadelphia, 1917). E. G. Janeway some years ago pointed out that when the viscera become involved during the tertiary stages of syphilis, there is apt to be loss of weight, moderate fever and night sweats. A careful examination will often reveal some other evidence of syphilis, such as enlargement and tenderness of ribs, clavicle or tibia, or an enlarged and indurated testicle. If the lungs are involved there is cough, which may be dry or accompanied by a moderate amount of greenish or yellowish sputum. Blood-streaked sputum may also occur. Pain at the base of the right lung may also occur and may be misinterpreted; it is usually due to perihepatitis and not to pleuritic inflammation. The presence of latent syphilis of the lung is to be suspected if, in addition to pulmonary symptoms, there are present elsewhere in the body lesions which are probably luetic, *e.g.*, periostitis, orchitis, iritis or suspicious throat lesions. The type of the disease which offers the most difficulty, is that in which the symptoms are entirely pulmonary and in which there are no associated syphilitic lesions. The five cases reported were of this type. In two cases the authors were unable at any time to detect syphilitic lesions in other parts of the body. In the other three the clinical picture at first was that of tuberculosis, but the subsequent appearance of aortitis (Case 3), of nocturnal headaches (Case 4), of an osteoperiostitis (Case 5), and the presence of a positive complement deviation reaction established the diagnosis. The symptoms were relieved as the result of anti-syphilitic treatment. The diagnosis must be made by exclusion. If the signs and symptoms are those of tuberculosis and the sputum does not contain tubercle bacilli, or the progress of the case differs from that usually encountered in tuberculosis, the possibility of some other cause should be thought of. The sputum should be examined for organisms other than tubercle bacilli and a complement deviation test should be employed, if necessary. In four of the five cases described the administration of salvarsan was followed by almost immediate relief. The fifth patient had not been seen since the administration of that drug.

(146) Tertian Malaria Contracted in France.

H. A. Cookson and T. S. Allen record a case of tertian malaria in a private who had never at any time been further east than France (*Journ. Royal Army Med. Corps*, April, 1917). In April and June, 1915, the patient had been in Flanders, near an Indian Division, and he was at that time much bitten by midges. He was admitted to hospital on March 27, 1916, with a temperature of 39.4° C., a pulse-rate of 104, and respiratory rate of 28. For some months he had not felt in good health, having been frequently feverish with shivering attacks. He had been acutely ill for eight days, and complained of pain in the left side of the chest posteriorly and also below the right infracostal margin. He looked pinched and debilitated. Incidentally there was found transposition of thoracic and abdominal viscera (heart, stomach, caecum, liver, etc.). The temperature was of the typical tertian character, reaching 39.4°, or 40° C., on each alternate day, the intervening days being apyrexial. A blood examination showed: red blood corpuscles, 4,500,000; white blood corpuscles, 7,000. hæmoglobin, 70. The red blood cells showed enlargement, poikilocytosis, vacuolation of protoplasm, pigmentation, and the presence of the parasite of tertian malaria. The patient was placed on quinine, and thereafter his temperature remained normal.

NEUROLOGY.

(147) A New Familial Infantile Form of Brain Sclerosis.

Knud Krabbe (*Brain*, xxxix.) recognized three types of diffuse sclerosis of the brain in children: (1) a syphilitic form; (2) Schilder's *encephalitis periaxialis diffusa*; (3) a familial infantile form. Of the last, five cases were discussed in this paper. Clinically, it seemed to be a family disease; it showed itself, somewhat acutely, at about the fifth month of life, in a child who had been previously healthy, and it terminated in death some five or six months later. Universal muscular rigidity, violent toxic spasms, probably painful, and brought on by touch or noise, were characteristic symptoms. As a rule, there was nystagmus, and, in the later stages, atrophy of the optic nerve. Causeless periodic elevation of temperature occurred. Extensive paresis and pronounced debility closed the scene. The pathological findings were induration of white substance without change of brain form. Microscopic examination of three cases showed practically complete destruction of the medullary sheaths and axis cylinders in the white substance throughout the nervous system, as against relatively intact cortex, basal ganglia and nuclear substance. The destroyed tissue was replaced by glial elements, the blood-vessels were surrounded by fatty granule cells and scavenger cells, and there was a complete absence of newly-formed vessels and of plasma-cells, lymphocytes and leucocytes. The disease was regarded as purely degenerative and non-inflammatory, it presented a certain relationship to Pelizaeus-Merzbacher's disease (*aplasia axialis extracorticalis congenita*), as well as to Tay-Sach's familial amaurotic idiocy, and yet differed conspicuously from both. (c.f. Abstract No. 66, *The Medical Journal of Australia*, August 19, 1916, p. 143).

(148) The Treatment of General Paresis.

Ogilvie (*Amer. Journ. of Syphilis*, July, 1917), in the treatment of general paresis strongly recommended the intraspinal injection of salvarsanized human serum, of standard strength, prepared *in vitro*, along with salvarsan given intravenously and mercury intramuscularly. Details of making the *in vitro* preparation must be read in the original. The intraspinal dose was 0.2 or 0.3 mg. of salvarsan in 10 c.cm. of serum. Injections were made at intervals of one week or ten days, for three or four treatments, and then intermitted for several weeks and recontinued according to the physical state of the patient, the character of the reactions and the general progress of the case. Combating the argument that the actual salvarsan content of a serum given intraspinally was necessarily so small that its value was questionable, Ogilvie pointed out that the action of strychnine given intraspinally was ten times more vigorous than when given intravenously. He added that salvarsan given *per se* intraspinally was dangerous; more than that, it was useless, because it was only spirochaeticidal when combined with blood serum. In 3500 injections by this method he had seen no untoward effect. Coming to results, he reported 55 cases (35 previously reported); in 18, there had been "complete remissions," of an average duration of two years and nine monts; in 23, "incomplete remissions," and in 14 failure to affect the course of the disease. He considered that intraspinal therapy combined with the other agents at hand, was of value in holding general paresis in abeyance, for a period dependent upon the extent of its activity at the time treatment was begun. To cure the disease when atrophic parenchymatous degeneration had set in was to expect too much. The real problem was to recognize invasion of the central nervous system at the very earliest stage. If this could be done general paresis might become a rare clinical picture.

(149) Occlusion of the Aqueduct of Sylvius in Hydrocephalus.

Schlapp and Gere (*Amer. Journ. Dis. of Children*, Vol. 13, No. 6, 1917) drew attention to the frequency of an obliterative lesion of the Sylvian aqueduct in hydrocephalus. They reported eight cases, all of which showed either complete closure of the aqueduct or obliteration of the fourth ventricle. The lesions were illustrated by microphotographs of sections which also served to show that without careful examination these changes might easily be missed. It was thought reasonable to ascribe not all but many, even the majority of cases of the congenital form of hydrocephalus to such a lesion. The closure of the aqueduct was usually due to a pericanalicular gliosis, concerning the origin of which they reasoned thus: in cells not highly specialized, such as the ependymal and glial cells, formative activity was easily awakened, hence stimulation of these cells by some irritant would lead to proliferation, and in a narrow channel like the aqueduct of Sylvius would soon cause closure. A case of hydrocephalus developing rapidly in a previous healthy child or adult might be due to such an irritative closure, but this ætiology was not applicable to the group of cases of hydrocephalus following meningitis, acute infectious diseases, and bacterial invasions of the brain by suppurative processes.

(150) Mercurial Treatment in General Paralysis.

Gaucher (*Ann. des Malad. Vénér.*, 1916, XLI.) protested against the view that mercurial treatment in general paralysis was useless, and, if prolonged, dangerous, on the following grounds. General paralysis was always a syphilitic affection of the tertiary stage, syphilitic alike in origin and nature. Mercury and iodides, especially the former, were always indicated in every case of general paralysis. Mercury never did harm, and might be always of service; it should be started as soon as possible. The treatment was often slow in effect, and therefore should be persisted in for years. Even when the treatment had produced its effect, and the disease appeared to be improved, if not cured, mercury should be continued indefinitely, at intervals, to prevent relapses. Gaucher recorded two illustrative cases; in one the patient had been seen by Brissaud, Raymond and Joffroy, who had all agreed in the diagnosis of general paralysis, and in the prognosis of incurability and an early death. These three eminent neurologists had all died, while the patient whom they had condemned, was still alive and in good health, and had been following a mercurial treatment for the past ten years.

(151) X-Rays in the Localization of Brain Tumours.

Heuer and Dandy (*Bull. Johns Hopkins Hosp.*, XXVI., 1916), from an X-ray examination of 100 cases of tumour of the brain, found that they might be divided into three classes, those which cast a shadow, those which showed some deformity of the skull, and those which radiographically gave no evidence of their presence. Only true bony tumours, or those containing lime, cast shadows which could be readily recognized. These were infrequent—about 6%. An uncalcified tumour might cast a shadow, if it could be brought into contrast with an air-containing space, such as the sphenoidal sinus. The changes in the form of the skull noted were those arising from increased intracranial pressure, namely, general enlargement and separation of sutures; and local changes, as arose in cases of pituitary tumour. In about 45 of the cases in the series X-rays had been of diagnostic value. The text was illustrated by a selection of well-printed radiographs.

(152) Diet in the Causation of Mental Disorder.

Mercier (*Journ. Ment. Science*, 1916, LXII.) described the symptoms in 35 cases of mental disorder, as well as the diet taken by each individual prior to treatment. The peculiarities in diet which preceded and accompanied the mental disorder in these patients were chiefly of two kinds, deficiency of meat, or excess of fat, starch or sugar. Rectification of the diet was followed by recovery in 94% of the cases. Excess of fat caused headache and sometimes mental confusion. Deficiency of meat brought mental confusion and depression—reaching in some cases to contemplation of suicide—and screaming fits, motiveless weeping, impairment of memory and hallucinations. Reasoning upon these facts, Mercier concluded that in a certain number of cases, small in proportion to the whole, but considerable in the aggregate, mental disorder was due to error in diet, and could be cured by correcting the error.

British Medical Association News.

SCIENTIFIC.

A meeting of the Queensland Branch was held on October 5, 1917, at the B.M.A. Room, Adelaide Street, Brisbane, Dr. W. N. Robertson, the President, in the chair.

Dr. L. N. McKillop showed a breast which had been amputated by the Hampson-Handley method. The operation had been undertaken for carcinoma.

Dr. J. Lockhart Gibson exhibited a skiagram showing a fragment of steel in the eyeball.

Dr. T. H. R. Mathewson referred to a series of cases of encephalitis which had formed the subject of a communication to the Branch at a previous meeting. He also read a pathological report by Dr. Oliver Latham on the macro- and microscopical appearances of the brain and the upper part of the spinal cord in one of the cases. We publish the paper, together with Dr. Oliver Latham's report, on page 352 of this issue. Dr. Mathewson expressed his indebtedness to Professor D. A. Welsh and to Dr. Latham for their valuable assistance in examining and reporting on the material sent to them.

Dr. W. N. Robertson thanked Dr. Mathewson and Dr. Latham for their exhaustive scientific report.

Dr. J. Lockhart Gibson congratulated Dr. Mathewson on the thorough way in which he had worked up his cases.

Dr. A. Stewart said that he had seen a case somewhat resembling Dr. Mathewson's, which was followed by an epidemic of acute anterior poliomyelitis.

A discussion followed on a paper by Captain H. Hunter Griffith on "The Clinical Treatment of Syphilis in the Australian Army." The paper was published in our issues of September 8 and September 15, 1917, and the discussion had been adjourned, pending the publication of the paper (see *The Medical Journal of Australia*, September 1, 1917, page 192).

Dr. W. F. Taylor said that the opinion he had come to after reading the paper was that the use of injections of arsenical preparations was attended by some risk to the patient. He thought that these injections should not be made by a general practitioner, but should be made only by an expert. The results obtained by treatment with arsenical preparations were no better than those obtained by mercurial treatment. He favoured the method of giving mercury for two years and iodide of potassium for two years. He had met with cases of optic atrophy and cerebral trouble which could be traced to treatment with arsenic.

Dr. G. H. Hopkins agreed with Dr. Taylor. He pointed out that Captain Griffiths had lost some of his patients. He, Dr. Hopkins, had worked under Sir Jonathan Hutchinson, who had not had a death under his method of treatment. He held the opinion that people generally found out what the patient was suffering from when he was confined to his bed. It was unnecessary for a patient to lay up during the course of mercurial treatment. The treatment could thus be carried out secretly. He saw no reason why mercurial treatment should be given up in favour of arsenical treatment. It was unnecessary for any deaths to occur as the result of the treatment of syphilis. It was their duty to persuade the patient to carry out the treatment conscientiously.

Dr. J. Lockhart Gibson held that if it could be shown that a few deaths under arsenical treatment would prevent more numerous deaths in the future, there was no doubt that the treatment did good. He had found that the use of mercury by inunction in eye cases was all he desired. He had never used the arsenical preparations. He did not think that iodide of potassium was much good in the treatment of syphilis, excepting in a few selected cases in the tertiary stage of the disease. He had listened with interest to Captain Griffith's paper, and thought it a pity that Dr. Molesworth had criticized it so severely.

Dr. J. Esp'e Dods congratulated Dr. Griffiths on his paper. Captain Griffith had realized the most important thing the military surgeon had to carry out. He had to get his patient well as quickly as possible and to send him back to his work. He did not agree with Dr. Taylor and Dr. Hopkins in regard to the treatment by arsenical preparations. He always treated prisoners with arsenic first, and then followed it up with treatment by mercury and iodide. He held that iodide was more than a tonic, and helped to clear up the symptoms when used with mercury.

Dr. L. M. McKillop had used intramuscular injections of *joha*, which was an oily emulsion of salvarsan. These injections were followed by the application of mercurial preparations. Since the organisms causing syphilis had been discovered, it was known that certain arsenical preparations exercised a great destructive action on them. There was no royal road to the cure of syphilis, but the quickest results were probably obtained by using both arsenic and mercury.

Dr. W. N. Robertson said that he still had certain suspicions concerning the new arsenical preparations, although they certainly had a magic effect in clearing up the symptoms quickly. He remembered having heard a paper read in Sydney in which the writer had said that, if necessary, he would give as many as 20 injections of salvarsan. He agreed with Dr. Gibson that Dr. Molesworth's criticisms were unkind. He was sure that they did not think any the less of Captain Griffith because of these criticisms.

Captain H. Hunter Griffith thanked Dr. Robertson for his kind remarks. He admitted inaccurate expression, but did not admit inaccurate observation. The inaccurate expressions had crept in owing to his having too short a time in which to prepare his paper. Dr. Molesworth had had exceptional opportunity for observation at the Royal Prince Alfred Hospital.

In reply to Dr. Taylor, he stated that in his opinion iodide had no specific action on syphilis, but that it assisted in breaking down the foci of fibrous tissue caused by syphilis, and in allowing the mercury to get at the organisms. Mercury was, of course, the mainstay of the treatment. The arsenical preparations were used to get a quick action, and were not used to cure the disease itself completely. The patients who had died under treatment by arsenobenzol, had been under care when they were still really experimenting with the drug. Now that it was given under better conditions, the reactions were very slight, and he thought that it was quite safe to give a full dose to start with. He considered it wise to give the men a typewritten sheet of instructions as to how to continue the treatment, as these men drifted about in different camps, and the medical officers with whom they came into contact were totally in the dark as to what treatment they had had. He had had no intention, when writing his paper, to pose as a syphilologist. He had set himself the task of describing the methods he had used in his attempt to get the man back to the lines as quickly as possible. He thanked the members for the kind manner in which they had listened to his paper.

The discussion on Dr. J. Lockhart Gibson's paper on "The Diagnosis, Prophylaxis and Treatment of Plumbic Ocular Neuritis Amongst Queensland Children" was resumed (see *The Medical Journal of Australia*, September 1, 1917, page 192, and September 8, 1917, page 201).

Dr. T. H. R. Mathewson stated that no one could be long in practice in Brisbane without realizing the prevalence of plumbism and of its unfortunate results. In dealing with the diagnosis he stated that no difficulty arose when there was a blue line and paralysis. He held, however, that it was possible to diagnose lead poisoning in the pre-paralytic stage. He considered that the examination of the gums for the presence of a blue line, and of the finger tips and nails, should form part of the routine examination of every child. He had looked up the records of 30 cases of plumbism treated at the Children's Hospital since December, 1916. The usual symptoms complained of were pains in the abdomen and limbs, and especially the legs, weakness in the limbs, paralysis, headache, vomiting, anorexia and constipation. The ages of the patients varied from 2½ to 11 years, 73% being under 8. In 15 of the patients there was definite paralysis of the muscles of the leg or arm. In two there was paralysis of the ocular muscle associated with optic neuritis, and in two there was optic neuritis alone. The urine had been examined by the Government analyst for lead in four cases. It was found in only one. The patient whose urine contained lead had a blue line, foot drop and punctate basophilia of the red blood cells. There was no increased pressure of the cerebro-spinal fluid. Lumbar puncture had been performed in 13 cases.

In seven the pressure was definitely increased, in four it was normal, while in two no record of the pressure was available. The paralysis could not be due to increased intra-cranial or intra-spinal pressure, as Dr. Gibson had suggested, at all events in all cases. A blue line was noticed in 10 of the 30 cases. Blood films were prepared in 22 cases. A punctate basophilia was noted 19 times, and nucleated red cells were found in two specimens. He held that this change was not due merely to an accompanying anæmia, for it occurred in cases where there was no corresponding reduction in the number of the red cells. The change had been noted in cases which could not be definitely proved to be cases of plumbism. He also referred to an increase in the resistance of red cells to hæmolysis, as evidenced by dropping blood into a hypertonic salt solution.

He did not consider it necessary to assume that the children got their lead poisoning exclusively by biting their nails or sucking their fingers. There were other ways in which the child could transfer the powdered paint to its mouth. He had seen plumbism in a child whose fingers showed no evidence of having been sucked, and whose nails had not been bitten.

He suspected that lead produced changes in the cardio-vascular system which were even more serious in their consequences than the changes in the eye. Lead had a very definite effect upon the arteries. It attacked first the intima, and later the media, and led to thickening of the vessels. This was followed by a chronic interstitial nephritis, increased blood pressure, cardiac hypertrophy and dilatation, and finally, heart failure. A Brisbane practitioner had told him that the patients treated by him for plumbism ten or fifteen years before, were turning up with chronic nephritis, and some with symptoms of heart failure.

In attempting to trace a relationship between plumbism and chronic interstitial nephritis, Dr. Mathewson pointed out that at the Children's Hospital 44 cases of plumbism were treated during the year, while at the Royal Alexandra Hospital for Children at Sydney there were no cases of this condition. Sydney had a population of children up to the age of 12 years of more than ten times that of Brisbane. He then turned his attention to the vital statistics. The death-rate from chronic nephritis for the age group 15-20 years for Brisbane was 9 times greater than that for Sydney. It was 5.7 times greater for the age group 20-25 years, and 9 times greater for the age group 25-30 years. In regard to the cause of these numerous cases, he expressed the opinion that many were not secondary to acute nephritis. Syphilis, alcohol and hard work could not be held responsible in these young people as in later life. He was inclined to attribute the cases to lead.

In conclusion, he dealt with prophylaxis. They did not know whether they had completely solved the problem of the causation of plumbism. But they did know that lead was present in a soluble form around them. Each one of them could do something toward diminishing the number of cases of plumbism by warning parents and children, while the school medical officers could do more by warning teachers and scholars. They should do all in their power to induce the Government to introduce legislation that would remove this very large and easily accessible source of lead. He thought that lumbar puncture should be carried out in all acute cases. Magnesium sulphate followed by iodide of potassium should be given. At Broken Hill an electric bath was employed in the treatment of lead poisoning. He understood that this treatment was very efficient in recent cases.

Dr. R. H. La B. Cummins said that in his student days, he had made many examinations of the urine for lead. He had been surprised to find how often lead was found in the urine. He was of opinion that the importance of using lead should be impressed upon the Government, in order that legislation prohibiting its use might be introduced.

Dr. E. W. Kerr Scott said that Dr. Lockhart Gibson should have all honour for discovering that lead poisoning was due to lead paint. He agreed that the Government should be approached again.

Dr. A. Stewart thanked Dr. Lockhart Gibson for his paper. His and Dr. Turner's work had obtained international recognition. He had pleasure in calling attention to a resumé of their work in an American journal. The best prophylaxis was not to use paint for the outside of houses at all, but to use boiled linseed oil without lead in its place.

Dr. W. N. Robertson said that they had more material to work on than previously, and that they ought to bring the subject before the Government again.

Dr. J. Lockhart Gibson stated that he had always considered that the lead poisoning was due to lead paint. It was not surprising that they got so many cases of lead poisoning, but it was surprising that they did not get more. The paper to which Dr. Stewart had alluded in an American journal contained a statement that cases had occurred through children eating the paint off their cot railings. He believed that zinc paint lasted longer than lead paint, although it was somewhat more expensive. More coats were required to cover the wood properly.

At the suggestion of Dr. Kerr Scott, it was resolved that a deputation from the Branch, together with Dr. J. I. Moore, the Commissioner of Public Health, should wait on the Home Secretary for the purpose of urging the Government to introduce a bill to protect the community from damage to health produced by lead poisoning.

A meeting of the South Australian Branch was held at the House of the Branch, Hindmarsh Square, Adelaide, on August 30, 1917.

Dr. B. Poulton exhibited two pathological specimens. The first was a caecum, which he had excised for carcinoma. The patient was a woman aged 60 years, who had complained of symptoms of dyspepsia and of pain for some months. There was a sudden aggravation of these symptoms before admission to hospital. There was an easily palpable mass felt above and to the inner side of McBurney's point. The condition closely resembled chronic appendicitis. The caecum had been removed together with the terminal portion of the ileum. The ileum and the ascending colon were closed, and later he had undertaken an anastomosis with the transverse colon. The patient had made a good recovery. The specimen showed invasion of all the coats but no ulceration. Two small glands, at least, in the meso-caecum were involved.

The second case was one of hydrocele of the spermatic cord. The condition had been present for about two years, and was attributed to a definite injury sustained while the patient was being dragged by a run-away cow. He had worn a truss with increasing discomfort. A small sac was present. It had been abolished at the operation.

Dr. H. Swift showed a man with an aneurysm of the arch of the aorta. He exhibited a skiagram of the chest. There were only two definite signs present. The first was a characteristic harsh cough, due to abductor paralysis. The second was d'Espine's sign as low as the 7th dorsal vertebra. Normally pectoriloquy could not be obtained below the 4th dorsal vertebra in adults or below the second in children.

Dr. R. Humphrey Marten demonstrated the case of an old man with marked symptoms of achondroplasia. The upper limbs were typical. The fingers of the right hand were of the same length. There was not such marked disproportion in the lower limbs. The head was not particularly large. The patient evidently had, besides his achondroplasia, very brittle bones, as he had fractured one humerus, one clavicle, one olecranon process, and one metacarpal bone.

Dr. R. Humphrey Marten read a paper on parasyphilis (see page 349).

MEDICO-POLITICAL.

A meeting of the New South Wales Branch was held at the B.M.A. Building, 30-34 Elizabeth Street, Sydney, on October 12, 1917, Dr. R. Gordon Craig, the President, in the chair.

Dr. R. H. Todd, the Honorary Secretary, moved on behalf of the Council:—

> That the following be adopted as Regulations of the Branch governing Position of Practitioner (Medical Examiner) examining Patient under care of another Practitioner (Medical Attendant):—
>
> (1) Except as hereinafter mentioned, the medical examiner should give the medical attendant such

notice of the date, time, and purpose of his visit as will afford reasonable opportunity for the medical attendant to be present should he or the patient so desire.

The exceptions are:—

(a) When circumstances justify a surprise visit.

(b) When circumstances necessitate a visit within a period which does not afford time for notification.

(c) When the medical examiner, after due enquiry made, has no information as to whether the patient is under medical care.

Where the medical examiner has availed himself of any of the above exceptions, it shall be his duty to inform the medical attendant, if any, as soon as possible, of the fact of his visit, and the reason for his action.

(2) The medical attendant must not put any unnecessary difficulties in the way of fixing a time convenient to both practitioners.

(3) If the medical attendant fails to appear at the time agreed upon, the medical examiner may proceed with his examination forthwith.

(4) The medical examiner must not, without the consent of the medical attendant, do anything in the course of his examination which involves active interference with the treatment of the case.

(5) Where the medical attendant fails to communicate with the medical examiner, the medical examiner shall, at his discretion and subject to the consent of the patient, make any examination he may consider necessary.

(6) The medical examiner must not make any comments to the patient which are of the nature of criticisms of, or reflections upon, the treatment, nor must he express, without the concurrence of the medical attendant, any opinion to the patient as to the ætiology, diagnosis, or prognosis of the case. His duty is strictly confined to examining into such matters as are necessary for the purpose of his report, and reporting to his employer, and to his employer only, his conclusions from such examination.

(7) If the medical examiner finds it necessary to report to his employer that any modification in the treatment which is being carried out, is in his opinion necessary to the more rapid recovery of the case, he shall so inform the medical attendant.

He pointed out that these regulations had been formulated by the Representative Body and the Council of the British Medical Association in Great Britain in 1911, and had been revised at the last meeting of the Representative Body. The necessity for these regulations arose on the Workmen's Compensation Act becoming law. The Acts passed in New South Wales and in Victoria had been based on wise if the same regulations were adopted in New South the English Act, and the Council thought that it would be Wales.

Dr. A. A. Palmer seconded the motion. A discussion ensued, in the course of which Dr. S. Sheldon raised the question as to whether these regulations should obtain in the case of a claimant under the Workmen's Compensation Act being brought to the doctor's consulting room. It was necessary for members to know whether they would be expected to refer the case to the medical attendant of the claimant, or whether they were entitled to give an opinion to the employer or insurance company.

Dr. Todd stated that these regulations did not include any rules covering the case of a claimant being brought by the employer or insurance company to a medical practitioner's room, but only applied in the case of visiting.

The motion was carried with one dissentient voice.

The following have been elected members of the New South Wales Branch:—

Arthur William Gordon, L.R.C.P., Lon., M.R.C.S., Eng., 1899, Auburn, New South Wales.

Hamilton Spier Kirkland, M.B., Ch.M., 1917, Univ. Sydney, Sydney Hospital, Sydney.

Raymond Arthur Dart, M.B., Ch.M., 1917. Univ. Sydney, M.Sc., 1915, Univ. Queensland, St. Andrew's College, Camperdown.

Edward Henry Stokes, M.B., Ch.M., 1917, Univ. Sydney, Wycombe Road, Neutral Bay.

David Leslie Howell, M.B., Ch.M., 1917, Univ. Sydney, South Sydney Hospital, Zetland.

The following have been nominated for membership of the New South Wales Branch:—

Reginald Francis Matters, M.B., Ch.M., 1917, Univ. Sydney, High Street, Unley Park, South Australia.

Roy Allen Sillar, M.B., 1917, Univ. Sydney, Royal North Shore Hospital, St. Leonards.

Special Correspondence.

(By Our Special Correspondent.)

LONDON LETTER.

King Edward's Hospital Fund.

The annual meeting of the Governors and General Council of King Edward's Hospital Fund for London was held at St. James's Palace on April 27, the Duke of Teck presiding.

Lord Revelstoke, the Honorary Treasurer, presented the accounts for 1916. It was a matter for congratulation, he said, that notwithstanding exceptionally heavy calls, the Fund had received such continued support as to enable it to put £143,000 to reserve. That was due mainly to the receipt of £137,000 from the residue of the estate of the late Lady Wilton. Since the end of the year a further sum had been received, raising the total of the bequest to £163,000. Had it not been for that legacy, and for other large legacies and gifts received in the past, the Fund would not have had the power it possessed to-day of affording such valuable assistance to the hospitals. The realization of the Fund's holding of American securities, which the Finance Committee considered it their duty to make in response to the invitation from the Treasury, and the reinvestment of the proceeds in British Government securities, had resulted in a small diminution in the income from investments.

The accounts were adopted on the motion of the speaker.

Mr. John Griffiths, Honorary Secretary, presented the draft report of the Council for the year 1916. It stated that the total receipts were £326,474, of which £9,447 represented contributions to capital, and £317,026 receipts on general account. The amount distributed was £170,000, which was £30,000 more than in 1915, and £12,500 more than the previous maximum appropriation before the war. Of that sum, £162,500 was allocated to London Hospitals, and £7,500 to consumption sanatoria and convalescent homes taking London patients, as against £133,500 and £6,500 respectively in 1915. Of the amount given to London hospitals, £128,425 was in aid of general maintenance, £15,000 for reduction of debts on maintenance account, and £19,075 towards improvement schemes, or in reduction of liabilities on such schemes when undertaken before the war. The increase in grants for maintenance over those made in 1915 amounted to £25,900. That large addition was, however, by no means in excess of the needs of the hospitals. The steady rise in prices of provisions and drugs, and in the cost of all departments, gave reason to fear that the needs of the hospitals had since continued, and would continue to increase; and those matters would demand the consideration of the Fund in future distributions. During the year the amount spent on administration was £3,161, or 19s. 4½d. per £100 of the total amount received, as compared with £3,205, or £1 8s. 3d. per £100 last year. The League of Mercy had contributed £15,000, a larger sum than was received from the League in any but the last three years. To maintain the annual distribution at £170,000, about £80,000 had to be raised each year from subscriptions, donations, and legacies. The subscriptions and donations received in 1916 were £31,440, an increase of £154 over 1915. With the addition of £15,000 from the League of Mercy, that source of revenue amounted

to a little over £46,000 out of the £80,000 required. The uncertainty attached to income from ordinary legacies was illustrated by the fact that, though in 1916 they amounted to £33,418, in 1915 they had only produced £4,412. The legacy from Lady Wilton thus provided a reserve against fluctuations in legacies during the coming years of possible stress.

Viscount Iveagh moved the adoption of the report, and Viscount Knutsford, in seconding the resolution, said they must all feel that it was very satisfactory that the Fund had been able to distribute £170,000 among the hospitals. The report pointed out that "the total additional ordinary expenditure of hospitals in 1915, after deducting War Office payments, as compared with ordinary expenditure in 1913, was over £48,000." That was only for 1915; but the Fund would have to face a much larger expenditure in 1916, and an increasing cost in the present year.

The report was adopted, and a vote of thanks was unanimously given to the Duke of Teck for presiding.

Hospital Expenditure.

The difficulties confronting great hospitals to-day are illustrated in some interesting figures concerning increased domestic expenditure, supplied by the Secretary of St. Thomas's. The meat bill has increased from £8,765 to £11,576, milk from £4,864 to £6,225, bread and flour from £1,947 to £3,397, grocery from £2,857 to £4,770. These figures speak for themselves of the urgent need for funds to keep the hospital going. The loss per out-patient has risen from 11½d. per attendance to 1s. 2d., and the cost of the in-patient department has risen from £77,374 to £88,299. The War Office pays 3s. per day for each soldier patient, and the actual cost to the hospital is 6s. 4½d. The cash shortage for the year is £5,165 15s. 7d. The secretaries of many of our largest hospitals complain that, with the advent of war charities, the public are inclined to overlook the needs of these old-established hospitals. There exists no greater need than that of the sick, the poor, and the wounded man.

The Causes of Still-Birth.

At a conference for the consideration of problems connected with maternity and child-welfare, recently held in Glasgow, Professor Munro Kerr read a paper of some interest on diseases and complications of pregnancy, and their relationship to still-births. In the course of his address, he emphasized the importance of the welding together of medical and social agencies in the struggle for securing a healthier world. Through notification, typhus, enteric fever, consumption, and other diseases had been reduced, and, through notification on lines such as he had previously indicated, he was confident that the complications of pregnancy would be greatly diminished. If the nation would insist that all women in pregnancy should be placed under favourable conditions and suitable supervision, the obstetricians, doctors, and nurses would reduce the number of still-births—the results of diseases of pregnancy—by somewhat about 90% and the death occurring by parturition by 65%, while in the process of doing that they would save thousands of mothers.

Correspondence.

BARCOO ROT AND BARCOO SICKNESS.

Sir,—Your issue of September 29, 1917, contains an interesting letter from Dr. Hill, of Laverton, on "Barcoo Rot," in which he also refers to the peculiar sickness known as Barcoo spew or Bellyander spew (a new name to me). My experience with this condition which was then simply known by the name of Barcoo sickness, has been limited to a trip I made into the interior of Australia in the early part of 1885. This trip was just after the heavy floods and rains that broke up the disastrous drought of 1883-4. It was not till we were beyond Innamincka, on Cooper's Creek, that we met with it. The malady usually follows very heavy rains, and many station managers and men think that the waters are infected by the countless hordes of flies that prevail then. Another view is that it is a form of malaria. A striking feature is that quinine is (or was) the most effective measure of relief. I saw a considerable number of cases, but was fortunate enough to escape being affected myself. My companion was not so fortunate.

Onset.—This is usually very sudden. A man may go into the dining quarters feeling quite well, and commence his meal with a good appetite. Suddenly an unsuppressible desire to vomit comes on, and a hurried retreat is made, and the stomach is emptied. Probably a return to table enables more food to be taken, and this is retained. Other cases may make two or more efforts, coming back each time with a keen appetite.

Others again find it difficult to retain anything, and rapid emaciation may result. There are no febrile symptoms and no other symptoms or signs beyond the utterly weary and tired feeling and the emaciation. As far as I know it is a local condition, affecting chiefly the region of the Barcoo River and some parts of the Diamantina River and Cooper's Creek districts. It disappears as the excessive moisture evaporates. One man I saw with it in a bad way, but some quinine and a little bismuth, which I had with me, enabled him to retain the first nourishment he had been able to keep down for some time. He then rapidly improved.

Barcoo Rot.—I do not think there is any relation between this condition and Barcoo sickness. It may follow the latter. Barcoo Rot is very like the ecthymatous sores seen in badly fed and ill-nourished children. I have seen numbers of cases in men from station country who have never been near the Barcoo country or had the sickness. It is the outcome of a long residence in a hot dry country on a diet of damper, Johnny cake, meat and tea without vegetables and milk. Sometimes the gums are swollen and bleed readily, suggesting a relationship to scurvy. Like Dr. Hill, I have always found some preparation of iron of the greatest value. Locally it is always essential to trim off the loose epidermis surrounding the sore, or the discharge burrows under it. The *ung. hydrarg. ox. rub.* and vaseline in equal parts ensures rapid healing, together of course with plenty of milk, butter and fresh vegetables.

Yours, etc.,

LEONARD W. BICKLE.

Sydney, October 17, 1917.

ROUTINE CIRCUMCISION.

Sir,—I am glad to see in your last issue some sensible remarks on the above subject from the pen of Dr. Sangster. The routine removal of a normal and healthy structure is surely the last word in absurdity. On rather rare occasions the prepuce needs removal, and should then be removed, but to make a practice of removing it is about as sensible as to make a practice of removing the labia minora or the little toe.

Circumcision was introduced as a religious ceremony—as an offering or sacrifice of part of the body. The Jews, with that abundant common-sense which still distinguishes them, chose the part of least value to themselves.

As Dr. Sangster remarks, there are "runs" or fashions in medicine and surgery, just as in women's hats, and they display about as much—or little—sense as the latter. To justify a religious ceremony on grounds of "cleanliness" or "morality" was quite an after-thought on the part of humanity, and merely indicates a tendency common in mankind to invent justifications for actions of which they do not understand the real significance, and also to use large, loose hazy words which really do not mean anything.

Yours, etc.,

ARTHUR S. VALLACK.

233 Macquarie Street, Sydney.
(Undated.)

Books Received.

GUM-NUT BABIES, words and pictures by May Gibbs; 1917. Sydney: Angus & Robertson, Ltd.; Royal 8vo., pp. 26. Price, 1s. net.

GUM-BLOSSOM BABIES, words and pictures by May Gibbs; 1917. Sydney: Angus & Robertson, Ltd.; Royal 8vo., pp. 24. Price, 1s. net.

THE INDIAN OPERATION OF COUCHING FOR CATARACT, INCORPORATING THE HUNTERIAN LECTURES, by Robert Henry Elliot, M.D., F.R.C.S., etc.; 1917. London: H. K. Lewis & Co., Ltd.; Royal 8vo., pp. 94, with 7 plates and other illustrations. Price, 7s. 6d. net.

ELECTRO-THERAPEUTICS FOR MILITARY HOSPITALS, by Wilfrid Garton, M.R.C.S., L.R.C.P.; 1917. London: H. K. Lewis & Co., Ltd.; Crown 8vo., pp. 48. Price, 2s. 6d. net.

HYGIENE AND PUBLIC HEALTH, by Louis C. Parkes, M.D., D.P.H., and Henry R. Kenwood, M.B., F.R.S., Edin., D.P.H.; Sixth Edition; 1917. London: H. K. Lewis & Co., Ltd.; Demy 8vo., pp. 787., illustrated. Price, 14s. net.

THE PRACTITIONER'S POCKET PHARMACOLOGY AND FORMULARY, by L. Freyberger, M.D., M.R.C.P., M.R.C.S.; 1917. London: William Heinemann; pocket size, pp. 545. Price, 12s. 6d. net.

THE IDEAL NURSE, An Address to Nurses, delivered to the Nursing Staff of The Retreat at York, at the opening of the Winter Session, 1909, by Charles A. Mercier, M.D., F.R.C.P., F.R.C.S., etc.; 1917. London: The Mental Culture Enterprise, High Holborn; pocket size, pp. 49. Price, 1s. 3d. net.

Medical Appointments.

The following appointments have been announced in the *Commonwealth of Australia Gazette* of October 18, 1917, in connexion with the Department of Trade and Customs:—

Dr. R. Macqueen (B.M.A.) as Quarantine Officer, Brisbane, from September 3, 1917.

Dr. A. R. Adams, as Quarantine Officer, Wyndham, Western Australia.

Dr. D. R. C. Tregonning, as Acting Quarantine Officer, Fremantle, during the absence on leave of Dr. Parer (B.M.A.).

Dr. Donald Albert Campbell (B.M.A.) has been appointed Acting Medical Superintendent of the Yarra Bend Hospital for the Insane.

Dr. Arthur Cardell Oliver will undertake the duties of Officer of Health in the town of Williamstown, Victoria, during the absence of Dr. H. R. Maclean (B.M.A.).

Dr. Charles Ellis Jelbart (B.M.A.) will act in the place of Dr. W. J. Forshaw (B.M.A.) while the latter is absent on active service, as Medical Officer of Health in the North and West Ridings of the Shire of Stawell, Victoria.

Dr. John Loftus Cuppaidge (B.M.A.) and Dr. Ernest Humphrey (B.M.A.) have been appointed Medical Referees for the purpose of "The Workers' Compensation Act, 1916," and of "The Workers' Compensation Act Amendment Act, 1916," for the whole State of Queensland.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xiii.

Royal North Shore Hospital, Sydney, Honorary Physician for Diseases of the Skin.

National Association for the Prevention of Consumption, etc., Honorary Assistant Physician at Anti-Tuberculosis Dispensary.

Medical Appointments.

IMPORTANT NOTICE.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429 Strand, London, W.C.

Branch.	APPOINTMENTS.
TASMANIA. (Hon. Sec., Belarive, Tasmania.)	Medical Officers in all State-aided Hospitals in Tasmania.
VICTORIA. (Hon. Sec., Medical Society Hall, East Melbourne.)	Brunswick Medical Institute. Bendigo Medical Institute. Prahran United F.S. Dispensary. Australian Prudential Association Proprietary, Limited. National Provident Association. Life Insurance Company of Australia, Limited. Mutual National Provident Club.
QUEENSLAND. (Hon. Sec., B.M.A. Building, Adelaide Street, Brisbane.)	Medical Officers to the Selwyn Hospital, North Queensland. Brisbane United Friendly Society Institute.
SOUTH AUSTRALIA. (Hon. Sec., 3 North Terrace, Adelaide.)	The F.S. Medical Assoc., Incorp., Adelaide.
WESTERN AUSTRALIA. (Hon. Sec., Health Department, Perth.)	All Contract Practice Appointments in Western Australia.
NEW SOUTH WALES. (Hon. Sec., 30-34 Elizabeth Street, Sydney.)	Australian Natives' Association. Balmain United F.S. Dispensary. Canterbury United F.S. Dispensary. Leichhardt and Petersham Dispensary. M.U. Oddfellows' Med. Inst., Elizabeth Street, Sydney. Marrickville United F.S. Dispensary. N.S.W. Ambulance Association and Transport Brigade. North Sydney United F.S. People's Prudential Benefit Society. Phœnix Mutual Provident Society. F.S. Lodges at Casino. F.S. Lodges at Lithgow. F.S. Lodges at Parramatta, Penrith, Auburn and Lidcombe. Newcastle Collieries — Killingworth, Seaham Nos. 1 and 2, West Wallsend.
NEW ZEALAND: WELLINGTON DIVISION. (Hon. Sec., Wellington.)	Friendly Society Lodges, Wellington, N.Z.

Diary for the Month.

Oct. 30.—N.S.W. Branch, B.M.A., Medical Politics Committee; Organization and Science Committee.
Oct. 31.—Vict. Branch, B.M.A., Council.
Nov. 2.—Q. Branch, B.M.A., Branch.
Nov. 3.—Vict. Branch, B.M.A., Nomination Papers for Election of Members of Council Issued.
Nov. 9.—S. Aust. Branch, B.M.A., Council.
Nov. 9.—N.S.W. Branch, B.M.A., Clinical.
Nov. 13.—Tas. Branch, B.M.A., Council and Branch.
Nov. 13.—N.S.W. Branch, B.M.A., Ethics Committee.
Nov. 14.—Vict. Branch, B.M.A., Branch.
Nov. 14.—North-Eastern Med. Assoc. (N.S.W.).
Nov. 14.—Vict. Branch, B.M.A., Nomination Papers for Election of Members of Council Returned.
Nov. 15.—Vict. Branch, B.M.A., Council.
Nov. 20.—N.S.W. Branch, B.M.A., Executive and Finance Committee.
Nov. 21.—W. Aust. Branch, B.M.A., Branch.
Nov. 21.—Western Suburbs Med. Assoc. (N.S.W.).
Nov. 23.—Q. Branch, B.M.A., Council.
Nov. 27.—N.S.W. Branch, B.M.A., Medical Politics Committee; Organization and Science Committee.

EDITORIAL NOTICES.

Manuscripts forwarded to the office of this Journal cannot under any circumstances be returned.

Original articles forwarded for publication are understood to be offered to *The Medical Journal of Australia* alone, unless the contrary be stated.

All communications should be addressed to "The Editor," *The Medical Journal of Australia*, B.M.A. Building, 30-34 Elizabeth Street, Sydney, New South Wales.